The American Journal of Surgery

is the leading independent surgical Journal. It publishes many papers read before the outstanding Surgical Societies, but it is *not* "the official organ" of any organization. Every manuscript is selected by the editors, as worthy of publication—nothing is published because "it was read at the meeting."

Copyright, 1937, by The American Journal of Surgery, Inc.

NEW SERIES, VOL. XXXVIII NOVEMBER, 1937 No. 2

EDITORIAL BOARD

Editor:

Thurston Scott Welton

New York

D. C. Balfour *Rochester, Minn.*
Carl Beck *Chicago*
George R. Brighton *New York*
Meredith F. Campbell *New York*
James T. Case *Chicago*
Isidore Cohn *New Orleans*
Bradley L. Coley *New York*
F. A. Coller *Ann Arbor*
George W. Crile *Cleveland*
E. C. Cutler *Boston*
R. V. Day *Los Angeles*
Charles A. Elsberg *New York*
Herbert C. Fett *Brooklyn*
C. R. G. Forrester *Chicago*
H. Dawson Furniss *New York*
J. H. Gibbon *Philadelphia*
Emil Goetsch *Brooklyn*
Charles A. Gordon *Brooklyn*
Donald Guthrie *Sayre, Pa.*
A. E. Hertzler *Kansas City*
Louis J. Hirschman *Detroit*
James M. Hitzrot *New York*
Frederick C. Holden *New York*
Emile F. Holman *San Francisco*
J. A. Hyams *New York*
R. H. Jackson *Madison*
John E. Jennings *Brooklyn*
W. L. Keller *Washington, D.C.*
H. A. Kelly *Baltimore*
T. J. Kirwin *New York*
Arthur Krida *New York*
A. V. S. Lambert *New York*
Maurice Lenz *New York*
H. H. M. Lyle *New York*
Jerome M. Lynch *New York*
Urban Maes *New Orleans*
Harrison S. Martland *Newark*
Rudolph Matas *New Orleans*
Roy D. McClure *Detroit*
H. C. Naffziger *San Francisco*
C. R. O'Crowley *Newark*
Louis E. Phaneuf *Boston*
Eugene H. Pool *New York*
Douglas Quick *New York*
N. P. Rathbun *Brooklyn*
H. A. Royster *Raleigh*
Henry S. Ruth *Philadelphia*
A. C. Scott *Temple*
M. G. Seelig *St. Louis*
J. Bentley Squier *New York*
H. J. Stander *New York*
George W. Swift *Seattle*
H. L. Updegraff *Los Angeles*
Grant E. Ward *Baltimore*
J. H. Woolsey *San Francisco*

EDITORIAL

UNSUSPECTED SURGICAL EMERGENCIES

AT first glance it seems curiously unnecessary to propound a question as to the safety of a patient who develops an acute surgical condition while he is in the hospital under treatment for another disease. At first glance it would seem that he of all persons on earth would be likely to have his condition recognized without delay and treated without loss of time—that he should come through the ordeal safely if human skill and care could ever save life.

Actually the facts are far otherwise. I have reviewed no statistics, but I recall many instances. In one of my own wards this winter there was a woman under treatment for a second degree burn of the hand, received when she fell into the fire during an epileptic fit. On her twenty-second day of hospitalization, after the intern and resident had made their nightly rounds, she complained of abdominal pain. She was a woman of many complaints, and the nurse paid only cursory attention to this one. Later, when the complaints continued, the intern was called, but he adopted the same policy of indifference and it was not until some eight hours after the onset that the light dawned and it was realized that something was very seriously wrong. When the staff man finally came on the scene, he wasted no time in getting her to the operating room, and operation confirmed his diagnosis of mesenteric vascular occlusion, so extensive that massive resection of the small bowel was required. This is a rapidly progressive and highly fatal condition, and perhaps she would have died under any circumstances, but the fact remains that it developed while she was under treatment in the hospital, and that eight hours of valuable time were wasted.

In a recent analysis of a series of cases of intestinal obstruction, we turned up an illuminating instance of this same sort. A man of seventy-odd years died after an operation for volvulus of the sigmoid, an obvious condition, it would seem, if

ever there was one. Operation was delayed for more than twenty-four hours after the onset of symptoms, although he was in the hospital, under treatment, when they developed. But he had had an operation for cataracts three days before, and the development of intestinal obstruction during his post-operative course was no part of the plan. A few weeks ago I saw an autopsy on a tuberculous patient, whose clinical diagnosis was pulmonary and intestinal tuberculosis. And so autopsy proved. But it also proved that the immediate cause of death was a ruptured ulcer of the cecum, which had not even been suspected ante mortem, although post mortem review of the history enabled us to put a finger on almost the exact moment of perforation.

A study of a series of cases of acute appendicitis revealed three non-surgical deaths that carry their own lesson. One occurred in a patient admitted many months before for decompensated cardiac disease, for which she was receiving expert attention. But autopsy revealed a ruptured appendix with generalized peritonitis, which had not previously been suspected. The same unsuspected findings were revealed at autopsy in another patient who had been hospitalized for several years with pulmonary tuberculosis. The third patient lost her life from acute appendicitis because it developed during the puerperium and was not even considered as a diagnostic possibility until it was too late to save her.

I have read with considerable interest and profound unbelief the statements of various authors to the effect that peptic ulcers do not perforate or bleed while the patient is under treatment. In a recent study of peptic ulcer we personally found three fatal perforations and twelve fatal recurrent hemorrhages, all of which occurred while the patients were in the hospital and presumably responding favorably to supervised medical treatment. I recall a private patient who twice had almost lethal hemorrhages while he was in bed in a hospital, undergoing a strictly supervised Sippy regimen. Vale and Cameron reported personally five fatal cases of perforation in hospital and located two similar cases in the literature (Vale, C. F., and Cameron, D. A. Gastric and duodenal perforation during hospital treatment. *Ann. Surg.*, 103: 353–361, March, 1936). Devastating though the commentary be on our methods, I am in full accord with their conclusion that the patient who suffers a surgical accident while in the hospital, undergoing treatment and surrounded by all the resources of medical science, is actually more likely to lose his life than is the patient who is stricken in his home or on the street.

I have seen a patient under treatment for peptic ulcer develop acute appendicitis. I have seen a patient suffering from a presumably recurrent attack of gallstone colic die from acute pancreatitis. I know of three instances in which patients with chronic kidney disease lost their lives from unsuspected intercurrent empyema. I recently heard of a very narrow escape: A woman of seventy years of age developed a gangrenous appendix which went unrecognized for twenty-four hours by her family physician who, like her family, classified her complaint of abdominal pain merely as one of her usual lamentations; rigidity was absent, as it frequently is in patients of that age. The neurasthenic is particularly likely to have an acute condition overlooked, and before the physician labels her—it is usually her—complaint neurosis, it might be well for him to remember the late Lord Moynihan's comment that the definition of that word is frequently "I don't know."

I have gone to no trouble to collect these instances. They have come to my mind as I wrote. And I put them down for what they are worth, adding to them, however, certain general statements which it might pay us all to remember. Any preexisting medical disease is likely to be aggravated by the development of an acute surgical condition, and may entirely overshadow the more urgent pathology. Any patient in a hospital under treatment for one disease

is entirely capable of developing another. The mere presence of one condition offers no immunity whatsoever against the onset of another. Even a proven neurotic patient is susceptible to any acute surgical ill. And the solution of the problem? That I do not know, except that eternal vigilance is the price of the patient's safety—even in a hospital.

URBAN MAES.

Subscribers to THE AMERICAN JOURNAL OF SURGERY *visiting New York City are invited to make the office of the publishers (The American Journal of Surgery, Inc., 49 West 45th Street, New York) their headquarters. Mail, packages or bundles may be addressed in our care. Hotel reservations will gladly be made for those advising us in advance; kindly notify us in detail as to requirements and prices.*

ORIGINAL ARTICLES

MALIGNANCIES OF THE RECTUM AND RECTO-SIGMOID*

CHAS. GORDON HEYD, M.D., F.A.C.S.

Professor of Surgery, New York Post-Graduate Medical School, Columbia University

NEW YORK CITY

THE gastro-intestinal tract is divisible into three main portions, the fore, the middle and the hindgut. The middle gut embraces that portion of the gastro-intestinal tract from the ampulla of Vater to just beyond the hepatic flexure. This portion of the gut tube has largely the physiological function of absorption and may be considered as an extension of the small intestine.

If one studies malignant disease of the colon, he is impressed by the clinical fact that operability decreases as the localization of the malignancy is in the right or left half of the colon; furthermore, that operative mortality and curability diminish in like manner.

It is axiomatic of surgery for malignant disease that the younger the individual the more grave the prognosis. While young individuals have great biological powers for combating sepsis, they have not the same resistance against metastatic or cancerous invasion that is enjoyed by the senescent.

Malignancy of the rectum and recto-sigmoid may be studied by a consideration of the following factors:

1. The inherent activity of the cancer cells. There are four clinical varieties of malignant neoplasms of the rectum: (a) papilliferous carcinoma; (b) adenoid carcinoma; (c) colloid or mucoid carcinoma, and (d) melanotic carcinoma. The incidence and degree of malignancy varies from the papilliferous to the melanotic. The former is frequently obstructive, seldom gives rise to extramural metastases and has a high percentage of five year cures. The latter is rare, never obstructive, always metastasizes, and is usually incurable.

2. Duration of neoplasm. Rankin has stated that the presence of recognizable symptoms in his series of cases before operation was over ten months. Miles estimated that in the ampulla of the rectum it requires six months for a malignant growth to extend one-quarter of the circumference of the bowel. Every patient that I have examined with carcinoma of the rectum, either on a first visit or by consultation, has had indicative symptoms for a period longer than five months, and significant symptoms for approximately ten months.

3. The associated factors of general debility. The majority of carcinomata of the rectum occur from 50 to 60 years of age, and there is usually some degree of constitutional impairment—conditions such as chronic myocarditis, chronic nephritis, chronic hypertension. A few of the patients have had diabetes.

4. The co-existing factors that are the direct result of the malignant process—loss of weight, the impaired nutrition, the cachexia, the anemia and the loss of appetite, all the sequelae of malignancy and dependent upon the situation and growth of the neoplasm.

5. The complicating factors in the growth itself: (a) hemorrhage; (b) obstruction; (c) fixation; (d) infection of the bowel wall and perirectal tissues; (e) perforation; (f) abscess, and (g) peritonitis.

* Delivered before the Rochester Academy of Medicine, Rochester, N. Y., Thursday, October 5, 1936.

The anus, rectum and the terminal portion of the sigmoid—"the recto-sigmoid"—must be considered as an anatomical and surgical entity. The recto-sigmoid comprises roughly 10 to 11 cm., 5 cm. of which is above the peritoneal reflex of the cul-de-sac of Douglas and 5 cm. is retro-peritoneal and devoid of peritoneum. The midportion of the retro-sigmoid has an acute lateral deflection whose purpose, according to Mayo, is to deflect the evacuation of contents into the rectum. At this point there is a change in the type of rugae corresponding very much to the rugae of the anal canal. It is problematic whether there is a definite nodal segment here, yet physiologically the juncture of the recto-sigmoid with the ampullary portion of the rectum does exercise a semi-sphincteric function.

Rankin in reporting a study of the statistics of the Mayo Clinic definitely allocated neoplasms of the rectum and recto-sigmoid as follows: 67 per cent in the recto-sigmoid, 31 per cent in the ampulla of the rectum, and 2 per cent in the anal canal.

The finger, on rectal examination, can reach as high as the right valve of Houston and palpate all rectal neoplasms. Occasionally in the knee-chest position, with the patient straining, a recto-sigmoidal tumor can be felt. Practically all carcinomata of the rectum and recto-sigmoid may be diagnosticated by digital and proctoscopic examinations. Probably in no other field of surgical examination is it possible to obtain so much information so readily and with such accuracy as in rectal neoplasms by the use of the finger and a sigmoidoscope.

About 10 per cent of the cases of carcinoma of the large bowel are discovered accidentally during a laparotomy for gall-bladder disease, gastro-duodenal ulcer, or an appendix. Cancer of the large bowel is fourth in numerical frequency of malignancies, occupying about the same relative percentage as carcinoma of the stomach. Two-thirds of the cases of carcinoma of the large bowel will be within the recto-sigmoid and rectum, and 70 per cent of the organic lesions of the large bowel will be found to be malignant.

The beginning of a growth in the rectum will act as an irritant to bowel function and there will be an increase in the normal functions of that bowel. Secretion will be increased and motor function activated. The earlier effects of carcinoma will therefore be irritative. This will disturb the normal bowel rhythm of the individual. The physiological functions of the large bowel depend upon rhythm and there is a normal rhythm which is personal to the individual. The eliminative function of the bowel under ordinary circumstances follows a rather precise rhythm. This is modified obviously when the individual has been a confirmed and inveterate user of cathartics. One of the first symptoms of neoplasm of the rectum is the vague and not dramatic, change in the normal bowel function or bowel rhythm—frequent trips to the toilet to evacuate small amounts of loose, mucoid stool, frequently admixed with blood.

The symptoms of malignancy of the rectum occur in the following order: (1) change in bowel habit, (2) bleeding, and (3) pain. Bleeding and pain occurred in 48 per cent of the patients with carcinoma of the rectum or recto-sigmoid. Rankin states that the symptoms have existed eleven months prior to seeking advice, and that 20 per cent of the patients have had operation for hemorrhoids during the period of their symptoms. The presence of blood continuously for consecutive days or weeks, unless otherwise proved negative, is from a malignant lesion.

It is apparent that the moment any obstructive process appears in the gastro-intestinal tract, there ensues varying degrees of physical deterioration. Obstructive symptoms are subacute, chronic or acute, and however they vary in degree, they all result in impaired nutrition from the mechanical factors of bowel obstruction. An obstructive lesion need not be of such gross degree as to produce marked obstruction to fecal passage, but may be only

sufficient to cause hypertrophy, edema, increasing permeability of the bowel wall to bacteria and nodal or glandular hyperplasia and adenitis from infection.

In patients suspected of harboring a neoplasm of the colon, the tendency to prescribe routine gastro-intestinal *x*-ray examination is associated not only with danger but also with lack of diagnostic acumen. Accurate roentgenological diagnosis is obtained by an opaque enema and not by a gastrointestinal series. The giving of opaque mixtures by mouth for colon neoplasms is always attended by the grave possibility of producing an acute intestinal obstruction. Furthermore, the presence of a barium meal proximal to an obstructive lesion of the sigmoid results in the formation of large round masses, varying from 1 to 5 cm. in diameter. These masses are passed only with the greatest difficulty and in many cases they remain for days, if not weeks, proximal to the neoplasm. This mechanism, I believe, was in one patient the cause of death by producing an acute colonic obstruction following resection of the sigmoid for carcinoma. After what we had considered adequate bowel decompression by cecostomy, the resection and anastomosis were performed. During the operation it was necessary to "milk" many hard concretions, one or two almost the size of a billiard ball, out of the proximal colon before proceeding with the anastomosis. We realized at the time that not all were removed. The patient subsequently died of peritonitis from leakage, and at post-mortem there was found a ball-like mass of barium firmly impacted into the recently made anastomotic lumen with leakage into the peritoneal cavity.

Colostomy is not the treatment for carcinoma of the rectum. To give a patient a colostomy and to leave behind a continuing local growth, with fixation, increasing colonic permeability by bacteria, with necrosis, slough, absorption and with invasion of the bladder and prostate in the male, or vagina, cervix, broad ligament and bladder in the female, is an undesirable surgical accomplishment.

Our problem is indeed difficult when in large, well-known rectal and colon clinics only 50 to 60 per cent of patients presenting themselves with neoplasms of the colon and rectum are operable; and furthermore, when the patients have been cognizant of symptoms for at least an average of ten months, and 20 per cent of the patients with carcinoma of the rectum have been operated upon previously for hemorrhoids. In many cases, colostomy is the only surgical procedure that is possible. Colostomy is only a means of avoiding intestinal obstruction. It should not be considered in any other relationship. The philosophy behind removal plus colostomy is that irrespective as to recurrence of the growth by abdominal and liver metastases such portion of life as remains to these patients is a life of fair contentment. Jones makes the observation that in no case in which he has performed a removal of the growth plus colostomy have any of his patients attempted suicide. This is quite the reverse of what occurs in patients who have had a colostomy and retain of their tumor.

The proper surgical attitude is that every case of carcinoma of the rectum and rectosigmoid must have an artificial anus as part of the operative technique. The results of segmental resection with anastomosis—the intubation of the upper sigmoid into the rectum—are all associated with disturbing complications, with early recurrence and what is more important, recurrence in the same general area as the original tumor which provided the indication for the surgery. A colostomy is not intolerable. The control of the colostomy is entirely dependent upon the patient and not upon the type of operation.

An untreated case with carcinoma of the rectum lives approximately two years, in pain and misery. The final outlook for every carcinoma of the rectum not operated upon is at the most two years of suffering. The exact percentage of surgical

mortality seems to me to be a relatively secondary consideration. A colostomy does prevent intestinal obstruction; it does relieve the patient of many of the symptoms of rectal irritability and tenesmus. The patients gain weight after the colostomy, but the rectal tumor remains. This means local metastases in the peri-rectal tissues, broad ligaments and bladder, perforation, peri-rectal abscess, etc.—in brief, all the terrible sequelae of a death from cancerous invasion into the adjacent tissues.

It is generally true that as one descends from the cecum to the rectum, the cancer shows a greater tendency for glandular involvement. Metastatic involvement of the lymph glands is encountered more frequently in rectal carcinoma than in any other portion of the large bowel. Forty-six per cent of Rankin's cases had malignant lymph nodes in the resected specimen.

The abdomino-perineal operation devised by Miles is a complete excision of the tumor and all the gland bearing tissue and bowel en masse. The lymphatics of the rectum are (a) intramural, (b) intermediary, and (c) extramural. The intramural system consist of two networks, one beneath the mucous membrane and the other between the muscle layers. The intermediary system consists of a subserous network, where the rectum is covered by peritoneum, and a lymph sinus where the rectum is not covered by peritoneum. The extramural lymphatic system of the rectum has, according to Miles: (a) an upward zone of spread draining into the lymph glands that eventually terminate in the angle of divarication of the iliacs from the aorta; (b) the lateral zone of spread which extends laterally involving the lymphatics of the levator ani, the prostate in males, the cervix and broad ligament in females; (c) the downward zone of spread which extends toward the external sphincter, the lower fibers of the levator ani and finally metastasizes in the groin. In addition, a rectal cancer will spread always by direct continuity of tissue and rarely by emboli erupting into the rectal veins of the portal system.

Surgical intervention is always indicated in cancer of the rectum and recto-sigmoid. Of outstanding importance is the question of operability. How many patients as they present themselves are operable? Operability determines the type of surgery to be instituted, as well as the operative mortality and curability. Jones has predicated the treatment of carcinoma of the rectum by a query; "Shall we operate only upon those patients whom we think we can cure or shall we operate upon all patients whom we think we can make comfortable, mentally and physically for one or more years?" Wherever the percentage of operability rises, the mortality will also increase, as many border-line cases will be accepted for surgery which otherwise would not be operated upon. Paradoxically, by increasing the operability the hospital mortality will be increased but at the end of five years more patients will be alive and free from recurrence than when the operability is kept low. In institutions where there are colon and rectal clinics and a community somewhat "colon conscious" the operability should be well over 50 per cent.

It is a surgical axiom that relief from mechanical obstruction must be obtained either by medical means or decompression operations before resection is attempted. The bowel above the neoplasm of the rectum and recto-sigmoid is edematous, infiltrated, many times hypertrophied and is an unsafe tissue for sutures. A diseased bowel sutured proximal to an obstruction leaks in contradistinction to non-leakage in a normal bowel.

Pre-operative treatment will include high caloric diet, full water maintenance, non-residue diet, and administration of small doses of castor oil for forty-eight to ninety-six hours previous to operation; cleansing enema twice a day, using normal saline plus tincture of quassia, 31 to the quart, until the return is clear. The day prior to operation 4 c.c. of paregoric every four hours for five doses, and the usual pre-

operative morphine immediately before operation. It is highly important that the intestinal contents be solid at the time of operation, as nothing is so difficult to handle or so hazardous as fluid stools that in spite of every technical procedure escape. Hence the desirability of perfect intestinal rest for at least forty-eight hours before operation.

Cancer of the rectum and recto-sigmoid is always a surgical condition. The type of operation to be employed will rest upon a consideration of a number of associated findings: the age of the patient, the type and location of the neoplasm, the duration of the symptoms, the associated debility and anemia. The guiding principles in dealing with this lesion is the imperative necessity of a permanent colostomy and the relief of obstructive symptoms by medical and, or surgical decompression—cecostomy—before either a permanent colostomy or resection is attempted. The aim should be always to remove the tumor.

Further education of the public will do much to increase the percentage of patients diagnosed earlier and before marked debility has occurred. A rise in operability to 50 per cent or over will mean a large increase in five year cures.

There are four surgical procedures available: (1) colostomy with or without cecostomy; colostomy without resection will not require cecostomy and will be a palliative procedure, restricted to the definitely inoperable group of patients; and (2) colostomy with resection. The abdomino-perineal resection of Miles is the operation of choice. In fact it is the only operation that meets the surgical indications of removal of tumor, bowel and gland-bearing area en masse. It may be performed in two stages as a graded procedure, or at one stage. Competent surgical opinion varies as to the wisdom of the two stage or the one stage procedure. The consensus of most surgeons is toward the more frequent employment of the one stage procedure. With adequate decompressive measures, blood transfusion, overcoming hypohydration, and high caloric, forced feeding diets, much can be accomplished to make the patient safe for the one stage operation. In determining for or against the one stage operation, every patient must be considered individually as no fixed rules can be laid down; much will also depend upon the technical experience of the surgeon. (3) Colostomy plus the perineal operation of Mummery. This operation has a low mortality and can be applied to the very debilitated and aged patient. In the hands of its author it has a mortality of approximately 3 per cent, although his operability was only 20 per cent. It requires less trauma and less surgical exposure than the abdomino-perineal operation. It removes the tumor as a local entity but does not remove the glandular area in the upper portion of the sacral hollow or the mesocolon. (4) Posterior segmental resection without permanent colostomy, but with a preliminary decompression cecostomy. I have performed this operation a few times. It preserves the sphincter function of a normal rectum but has countervailing disadvantages. The excision is restricted in area and amount. The gland-bearing area is not removed. Posterior fistula and cicatricial contracture of the lumen with stenosis is frequent and recurrence almost always appears. I have discontinued its use.

SUMMARY

1. Consideration has been given to the physiology and anatomy of the large bowel and the distinction drawn between the right and left-sided colon.

2. The clinical portraiture of malignancy of either half of the colon is given.

3. Emphasis has been given to the ease of diagnosis of carcinoma of the rectum and recto-sigmoid by utilization of the palpatory finger, plus sigmoidoscopic examination.

4. The surgical axiom that rectal bleeding must be accurately allocated to its anatomical source is reiterated, with emphasis placed upon its diagnostic importance.

A NEW METHOD IN THE USE OF RADON GOLD SEEDS

FRED HAMES, M.D.*

NEW YORK CITY

THE use of radium in the treatment of malignancy has advanced continuously since the discovery of this element by the Curies, more particularly since means have been perfected for using emanation collected from radium in solution enclosed in capillaries of glass or gold. The use of the emanation or radon has greatly increased the flexibility of radiation, and the work herein described was carried out with a view to further increase its use and flexibility and provide a more simple means and, in some cases, a more efficacious one than possible with the usual application by platinum needles.

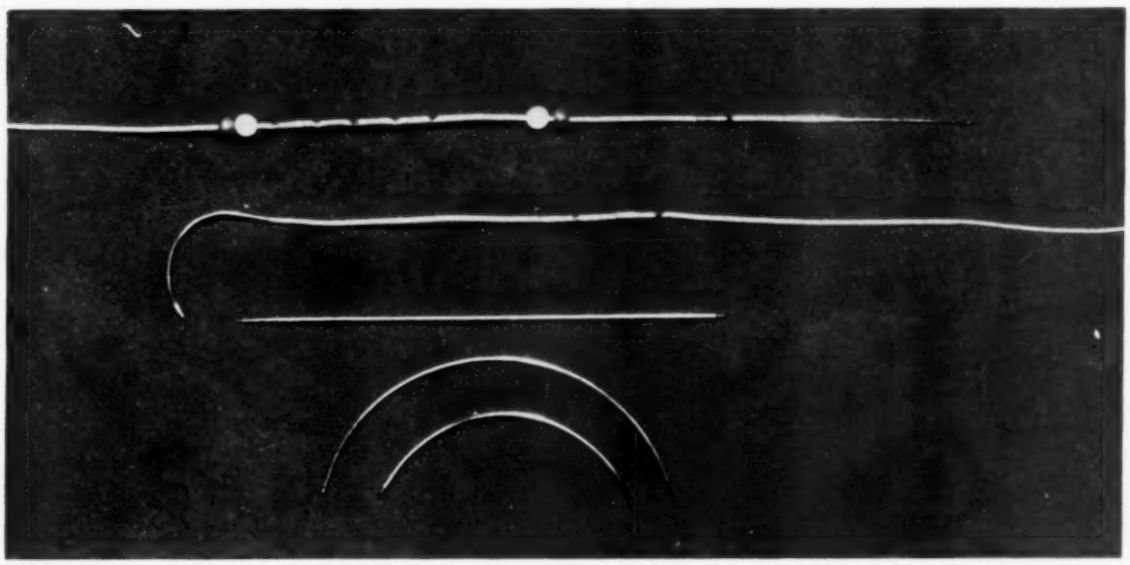


FIG. 1. Upper illustration shows needle and beads with anchoring shot. Also three needles of different types unattached.

In the use of needles containing radon or radium, the mobility of the involved part is greatly limited or temporarily destroyed. This presents a problem, especially in cases of malignancy of the tongue and other portions of the oral cavity. With platinum needles in situ, it is impossible for the individual to dispose of the saliva, etc., within his mouth, and pulmonary complications are not unusual as a result. Inaccuracy of the placement of needles in other portions of the body is occasioned by the lack of flexibility of the platinum needles. In an effort to overcome some of these objections and to increase the more general use of radon, a radon suture method was developed.

The method consists in placing of radon seeds within the lumen of a specially made silk carrier. The spacing between openings into which the radon is placed can be varied at will. Those used in the cases herein described were all placed 1 cm. apart. The usual filtration of .3 mm. of gold has been employed but this may be increased as desired. After the radon seeds are placed into the body of the woven silk material, they are permanently sealed in place by the closure of the woven structure and also by a plastic substance containing carmine as an indicator of the area in the

* New York City Cancer Institute.

suture in which the radon is situated. The spaces between the implants are filled with silk, so that there is a uniform diameter of the suture material throughout its length. On one end of the suture is a small, permanently attached metal collar. The distal end of this collar is threaded to receive the needle of whatever type may be desired. The needle is threaded at one end in order to permit its being screwed into the metal collar. This obviates the necessity of threading a needle and dragging a double portion through the tissue to be treated. The suture is used as follows: The needle is passed through the tissue around or beneath the malignancy and the proper placement of the suture guided by the red markings indicating the point of radon activity. After the suture is so placed, a glass bead is threaded over either end and brought to rest against the tissue. Above this bead a lead shot is fixed to hold the material in place until such time as its removal is desired. In removing the device the suture is cut below the fixed bead and withdrawn in a manner similar to that used in removing an ordinary skin suture or capillary drain.

A B C

FIG. 2A. Large ulcerating metastatic tumor of neck. B. Three sutures containing twenty-five seeds of 1.5 mc. each. Total dosage 2500 mc. hours. Number of hours in place, 120. C. Same case eight days after insertion showing radium reaction and reduction of ulcerated area.

D E

FIGS. 2D and 2E. Two views of radon seed in tumor of neck.

In such cases where it is unnecessary or impossible to use a needle, the radon loaded suture may be packed into the cavity—as the antrum, uterus, etc.—after being attached to the usual packing gauze, and its

A B

Fig. 3A. Malignancy of the mid-lateral portion tongue. 3B. Four sutures containing nineteen seeds of 2 mc. Total dosage 2000 mc. hours. Number of hours in place, 73.

Fig. 3C. Radiograph of tongue showing distribution of radon seeds.

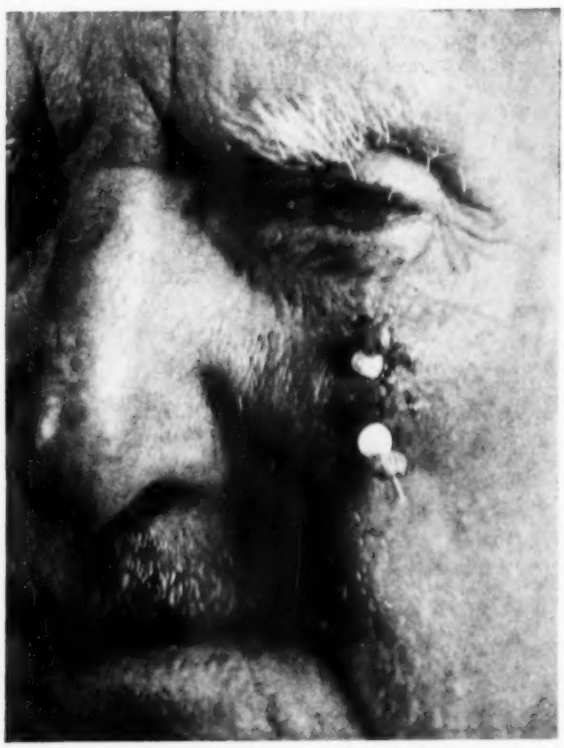


Fig. 4. Suture containing two gold seeds of .3 mc. each.

removal readily accomplished by removing the packing.

All of the material may be sterilized at the time of use, either in the autoclave, or by boiling, or by chemical sterilization not destructive to silk.

FIG. 5. Squamous cell epitheloma of lip with four sutures containing eleven gold seeds .5 mc. each. Total of 500 mc. hours. Number of hours in place, 150.

In the photographs several different types of needles attached to the radon suture are shown, and several cases in which the suture has been applied in malignant conditions are illustrated.

To conclude, this method is offered as an improved technique for employing radon seeds because of:

1. Simplicity
2. Accuracy
3. Patient's comfort
4. Elimination of secondary radiation
5. Reduction in nursing care, due to the fact that the majority of the patients treated are ambulatory
6. Permits increasing of the amount of fluid or food intake in mouth cases, and does not prevent the ability to expectorate
7. Multiple seeds (one to twenty) can be applied with one procedure
8. Provides a means of using radon by the average surgeon, who may be distant from the source of supply of radium and radon.

This method has been used at the New York City Cancer Institute (Dr. Ira I. Kaplan, Director). The author was assisted in the preparation of this method by the Director and by the staff of the Cancer Institute, and also by Mr. Hoffman and Mr. Cameron, technicians in charge of the radon plant, and by Mr. Carl B. Braestrop, physicist of the Institute, who made the required measurements, to all of whom his thanks are extended.

The needles described and illustrated were designed and perfected by J. A. Deknatel and Sons Inc., of Queens Village, L. I., N. Y., and their technical staff has rendered every possible assistance in the manufacturing of the silk material.

POST-OPERATIVE COMPLICATIONS FOLLOWING SUPRA-PUBIC PROSTATECTOMY AND THEIR PREVENTION*

M. MUSCHAT, M.D., F.A.C.S.

Associate in Urology, School of Medicine, University of Pennsylvania; Attending Urologist, Mt. Sinai Hospital

PHILADELPHIA, PENNSYLVANIA

THE old dictum "no prostatectomy without a complication" still holds true today. Many complications of yesterday are still with us today in spite of our greatly improved technique and increased knowledge of the pathological physiology in the prostatic gland.

The low mortality of 4 to 5 per cent in the leading clinics of today could never have been achieved without much research and painstaking care in an effort to anticipate and to prevent the postoperative complications. While the complications still occur, we know better today when to anticipate them and, if unavoidable, how to minimize their severity. Failure to combat the first signs of impending postoperative difficulty will frequently result in a grave irreparable morbidity or even mortality.

In this paper I intend to discuss the postoperative complications in properly selected and prepared patients.

COMPLICATIONS DUE TO ANESTHESIA

We prefer nitrous oxide oxygen as the safest anesthetic for an aged man. Administered competently with a short excitation stage, the patient leaves the operating room without any shock and a warm skin, without interfering with his cardiovascular system. If spinal anesthesia is given we always ask ourselves the question, "Will the cardiovascular system of this patient be able to withstand a sudden drop in blood pressure? Is it safe for this particular cardiovascular mechanism to administer ephedrine or adrenalin to raise a fallen blood pressure?" We believe that any anesthetic which tends to disturb the compensated balance of the vascular system may be injurious and the cause of much postoperative morbidity.

The preoperative administration of sedatives is not desirable and in many instances injurious. We are advised against the use of preoperative morphine and atropine by our bronchoscopist who feels that morphine, causing shallow respiration, and atropine, causing the stoppage of bronchial secretions with the subsequent thickening of the already present mucus, may be the cause of postoperative atelectasis. Atelectases are more frequent than is generally believed. An alert internist will often point out its presence in a mild degree without any subjective or clinical signs.

COMPLICATIONS DUE TO INFECTION

The sources of infection are numerous. It starts in the operating room. The patient should be placed on the table with both legs elevated and resting and a special sheet covering each leg to allow the surgeon or outside assistant enough sterile space between the legs for further procedures during the operation. The outside assistant should wear gloves and gown. Many surgeons still fill the bladder with water to facilitate suprapubic exposure. We have not done it for the past few years as we believe that the preoperative filling of the bladder, irrespective of the nature of the fluid used, is not without danger in an anesthetized person. The sudden uncontrolled increase in the intracystic pressure in an infected

* From the Departments of Urology, Mt. Sinai Hospital and the University of Pennsylvania Hospital, Philadelphia, Pa. Read before the Philadelphia Urological Society, November 23, 1936.

bladder may precipitate and disseminate ascending infection. Many renal infections observed to develop four or five days later have their origin in the overdistention of the bladder on the operating table. If care is taken the identification of the bladder by the suprapubic route presents very little difficulty. The second source of infection is the insertion of the finger into the rectum to facilitate the enucleation of the prostate. It should not be practiced routinely. If one must use this method the utmost care and caution in maintaining sterility should be observed. The entire forearm should be covered with a sterile sheet and pinned with a towel clip before undertaking it. The removal of this sheet and the changing of the glove should be done meticulously by a specially trained nurse.

The third source of infection lies in the method of introducing or removing the hemostatic bag. The passage of a curved metal catheter through an infected urethra not only traumatizes the prostatic bed but also carries the infection from the outside directly into the raw and vulnerable field of operation. A complete or incomplete false passage may easily be overlooked or pass unnoticed with subsequent perivesical suppuration. This type of diffuse perivesical suppuration is extremely difficult to diagnose, acting as a closed phlegmon in the depth of the pelvis and only found at autopsy. This method of bag introduction must be discarded. Our method of choice is as follows: (Fig. 1) After opening the bladder the intravesically projecting part of the catheter is grasped with a forceps; a long piece of catgut is sewn to the catheter tip and tied; the outside assistant then pulls the catheter with the catgut string sewn to it out through the urethra, leaving sufficient catgut on the operating field. One then proceeds with the enucleation, completely disregarding the catgut, since the operator does not feel the string with the fingers unless it is drawn taut. After completion of the enucleation a hemostatic bag is tied to the catgut string and pulled down by the outside assistant.

The removal of the bag, usually done forty-eight hours later, also demands sterile

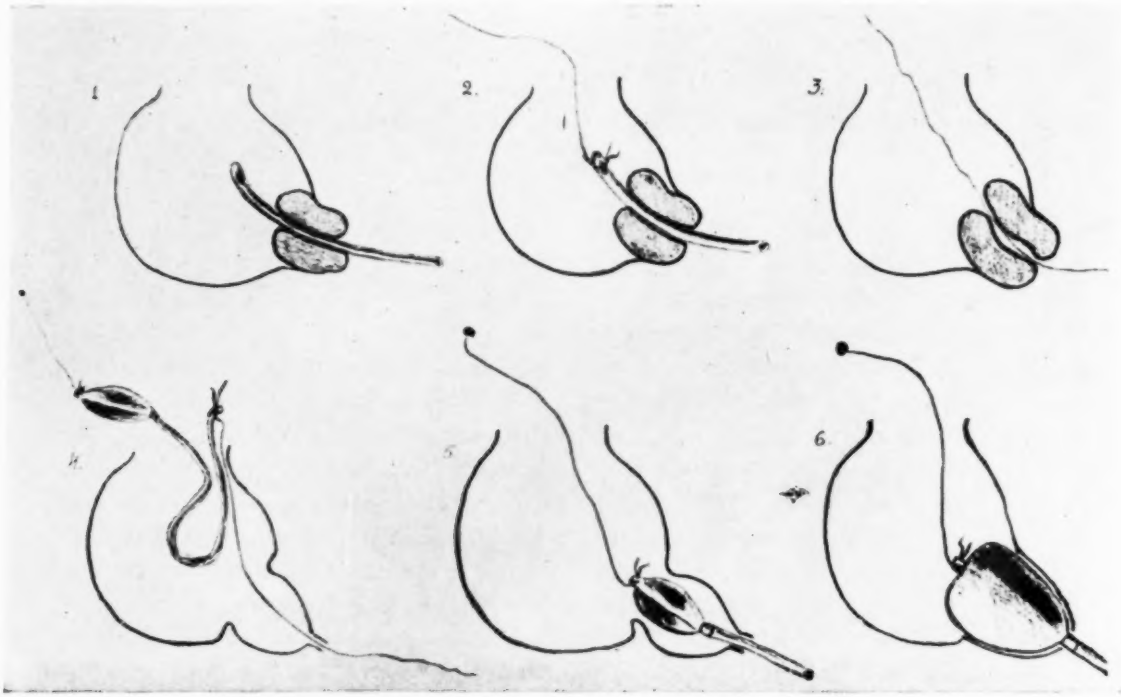


Fig. 1. Method of introduction of the Hagner bag.

precautions. The bag is pulled out through the wound to its neck and cut off. The urethral portion of the bag is then utilized to irrigate the bladder and wash out any accumulated clots or débris and is removed only after the entire dressing is completed.

The fourth instance of introducing infection is during the daily dressings. We insist on individual dressing trays and gauze drums, thus avoiding contamination.

COMPLICATIONS DUE TO OPERATIVE TECHNIQUE

In enucleating the gland one must be radical one way and conservative the other. One must be radical to remove all the obstructing tissue including the occasionally missed hypertrophied tissue within the commissure, otherwise one experiences delayed closure or actually has to resort to transurethral resection of the leftovers in the commissure. On the other hand, one must be conservative and cautious in shelling only within the line of cleavage, thus avoiding breaking off the partially enucleated gland with the resulting difficulty for complete removal of the prostatic adenoma. Such care during enucleation will also prevent postoperative incontinence of varied degree not infrequently seen after suprapubic prostatectomy.

In the closure of the bladder, we cover the first line of sutures with the opened pelvic fascia, taking a few extra bites through the bladder wall away from the suture line in order to invert or bury the first line of sutures. We believe it to be responsible for a great drop in perivesical infection on our service. For many years we have discontinued the drainage of the Retzius space and have not found it necessary to resort to it again.

In doing the cystotomy for the first stage cases we invariably anchor the dome of the bladder to the uppermost edge of the wound in order to insure sufficient extraperitoneal space for any subsequent operation, thus preventing opening of the peritoneum at the second stage.

Complications Immediately and during the First Twenty-four Hours Following Operation. In the very aged and decrepit, we insist on bringing the patient up to the operating room in his bed. During his stay in the operating room his bed is heated with hot water bottles and upon completion of the operation he is placed in a warm bed, avoiding unnecessary moving and any chilling of a sweat covered body.

We believe that a certain degree of shock is always present after prostatectomy and therefore upon return to his room the patient receives 1000 c.c. of 5 per cent glucose by hypodermoclysis and is stimulated every three hours with digitalis and caffeine. To alleviate the painful spasms from the hemostatic bag, small doses of morphia are administered. However, one is rarely able to control these spasms and it would be safer not to administer morphia at all. No p.r.n. order of morphia should ever be given immediately following prostatectomy.

If shock occurs it should be combated instantly by the regular means at our command, as intravenous infusion, large doses of digitalis and caffeine. Small doses of adrenalin (3 minims) at one-half hour intervals until proper restoration of circulation has been very useful in our hands. If the shock is prolonged blood transfusion is life-saving and resort to it should be without delay. Following the restoration of circulation one should continue treating the shock by continuous venoclysis for the next twenty-four hours as one encounters cases with a secondary circulatory collapse after an apparent improvement.

The next complication to consider is hemorrhage. One rarely sees nowadays any really serious bleeding due to the routine use of the hemostatic bag. We leave the bag in situ until the next morning believing it safer to have it in place during the night. However, hemorrhage may occur on the best managed service due to faulty placement of the bag, improper fastening on the outside, leakage of the water through a pin-point hole or tearing of the rubber tube

from too much tension. We once saw a patient who, after removing the Freyer tube and Hagner bag two hours after operation, got out of bed and started to the bathroom. Nothing untoward ever happened to him. Occasionally a patient overcome with pain will pull on the cage and release the pressure. A very devoted nurse or interne, unfamiliar with the mechanism of the hemostatic bag and believing the urethral tube in reality to be a catheter, will remove the hemostat to allow the urine to flow out without realizing the futility of such an attempt. If such an accident occurs it should properly be reported to the surgeon in charge as it is an emergency of grave importance. If the bag is intact it can easily be refilled with water and the hemorrhage controlled. One should always remove the dressings and remove all clots including the ones in the Freyer tube. When the bag is leaking, it is pulled out to its neck suprapubically and a new one tied to the cut end of the urethral portion of the old one and pulled into position. A bag torn off will have to be replaced by the gentle passage of a curved metal catheter through the urethra.

However, there is another type of hemorrhage which is not as spectacular and may go unnoticed for a long time, namely, the slow ooze. Such oozing is usually urethral and will not be noted unless the patient is uncovered and the bed examined. It is therefore very important to instruct the nurse to investigate the bed for bleeding every hour during the first night.

A slow hemorrhage of such character is usually controlled by filling the hemostatic bag with more water and bandaging the penis.

During the first twenty-four hours one also encounters instances of cardiac failure and attacks of angina pectoris. The prompt aid of the internist is often lifesaving. Many of these cardiac attacks are brought on by the severe painful spasms caused by the hemostatic bag. Heavy sedation is strongly indicated and should be started immediately after operation. It also seems wise in such instances to empty the bag to give the patient temporary relief, later gradually refilling it. Whenever we had to resort to this measure we were surprised to find very little or no bleeding.

One encounters atelectasis in the first twenty-four hours but usually it does not make itself subjectively felt until thirty-six to forty-eight hours after operation. Most of the patients with atelectasis are easily controlled by frequent change of position and the administration of a mixture of carbon dioxide and oxygen. A tight abdominal binder will greatly aid in reducing the apprehension to breathe deeply and to cough. It is imperative to establish the correct diagnosis of the lung involvement early, since the therapy is often diametrically opposite. In atelectasis one moves the patient into various positions, even sitting him up in a chair, while in true pneumonia such treatment would be contraindicated. Frequent films of the chest are taken to note improvement or aggravation of the condition. In some cases with very marked retraction of the heart and dyspnea bronchoscopic aspiration is indicated.

Complications within First Few Days after Operation. Gastric distention is frequent and is mainly due to ingestion of large quantities of cold water. Such a distention may be the initiating cause of cardiac failure and should therefore be considered a surgical emergency in an old man and immediately combated. Immediate institution of Wagensteen drainage will remove the distention, keeping it up for at least twelve hours. Gastric distention, even of a minor degree, may also be the cause of hiccough. We believe that hiccough is more frequently caused by gastric dysfunction than by toxemia. The Wagensteen technique instituted as soon as hiccough occurs will stop it in most instances. In very neurotic patients a single or repeated cocainization of the sphenopalatinal ganglion is very effective in stopping the hiccough. The distention of the bowel does not occur until thirty-six to forty-eight hours later and is prevented by

the early administration of castor oil on the night of the second day. The evacuation is greatly aided by prostigmine, pituitrin or a small glycerine enema given through a small soft rectal tube. One should not use stiff nozzles, insert rectal tubes or give a high enema as it occasionally may cause perforation of the rectum and bladder or stir up serious vesical bleeding.

The postoperative hyperpyrexia lasts only two or three days. However, if it persists one should look for the cause. Occasionally one sees an aged patient whose temperature is normal but on taking it rectally it is found to be elevated (Fig. 2).

The blood urea nitrogen during the first three days usually shows higher values. This is to be expected and should not be interpreted as impending uremia with the resulting heroic hypodermic administration of large amounts of water. Such unwarranted water intake may be very injurious especially when the cardiovascular system is damaged. It is also of great importance to determine the CO_2 value daily for the first four days postoperatively, thus detecting an acidosis early and enabling one to combat it by the intravenous administration of Fisher or Hartman solutions followed up with alkalis by mouth, before it assumes serious proportions.

If the temperature persists a careful investigation of the wound must be undertaken. Stitch abscesses or breaking down of the wound should receive prompt attention. Perivesical infection is the most frequent complication, causing partial or complete breaking down of the wound. The best treatment is the removal of the stitches and wound toilet several times a day using only mild antiseptics preferably peroxide, followed by balsam of Peru packing. Irrigation of the bladder per urethra is very valuable to prevent stagnation in the base of the bladder.

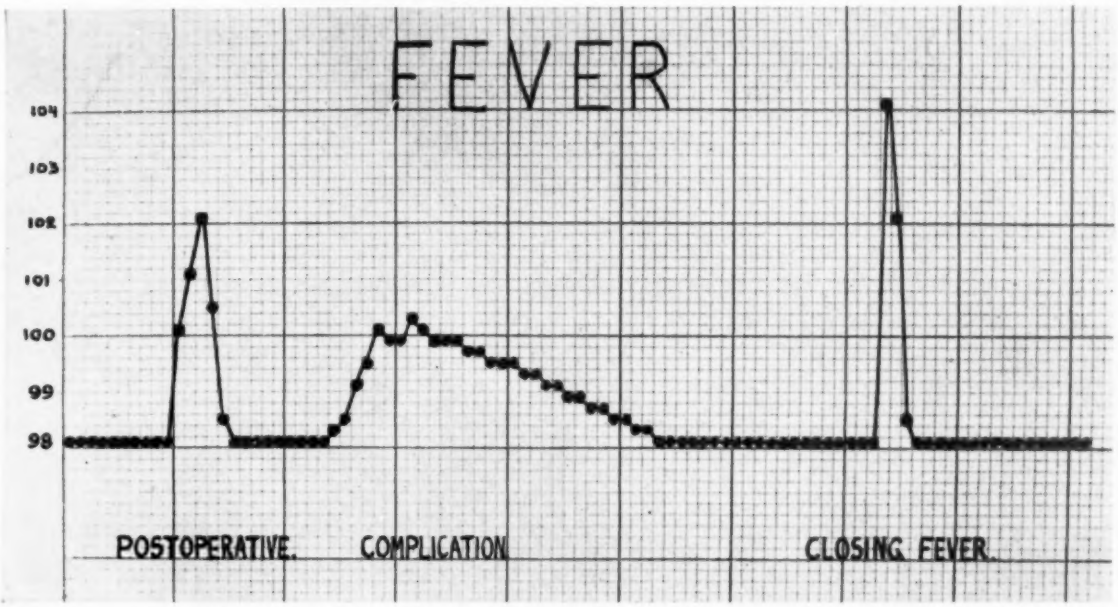


Fig. 2. Fever chart, illustrating the post-operative fever, the fever due to complications and the closing fever.

Another complication worthy of consideration is the phosphatic incrustation of the wound seen on the third or fourth day. We found the use of 0.5 per cent phosphoric acid irrigation as advised by Randall to be a specific and notice the reappearance of the red granulations within twenty-four to forty-eight hours.

In some cases painful hemorrhagic urination with chills and fever is initiated on the third or fourth day. This should be immediately combated by a suprapubic drain. Occasionally a fistula becomes covered by

the rapidly growing skin and stops the urinary flow. Reopening with a probe and drainage with rubber dam placed all the way into the bladder is indicated to prevent bacteremia due to a premature increase in the intracystic pressure. Any insertion of drainage into the fistula should always be done cautiously and never be forced. Cases have been reported in the literature where a suprapubic tube was thus forced into the peritoneal cavity.

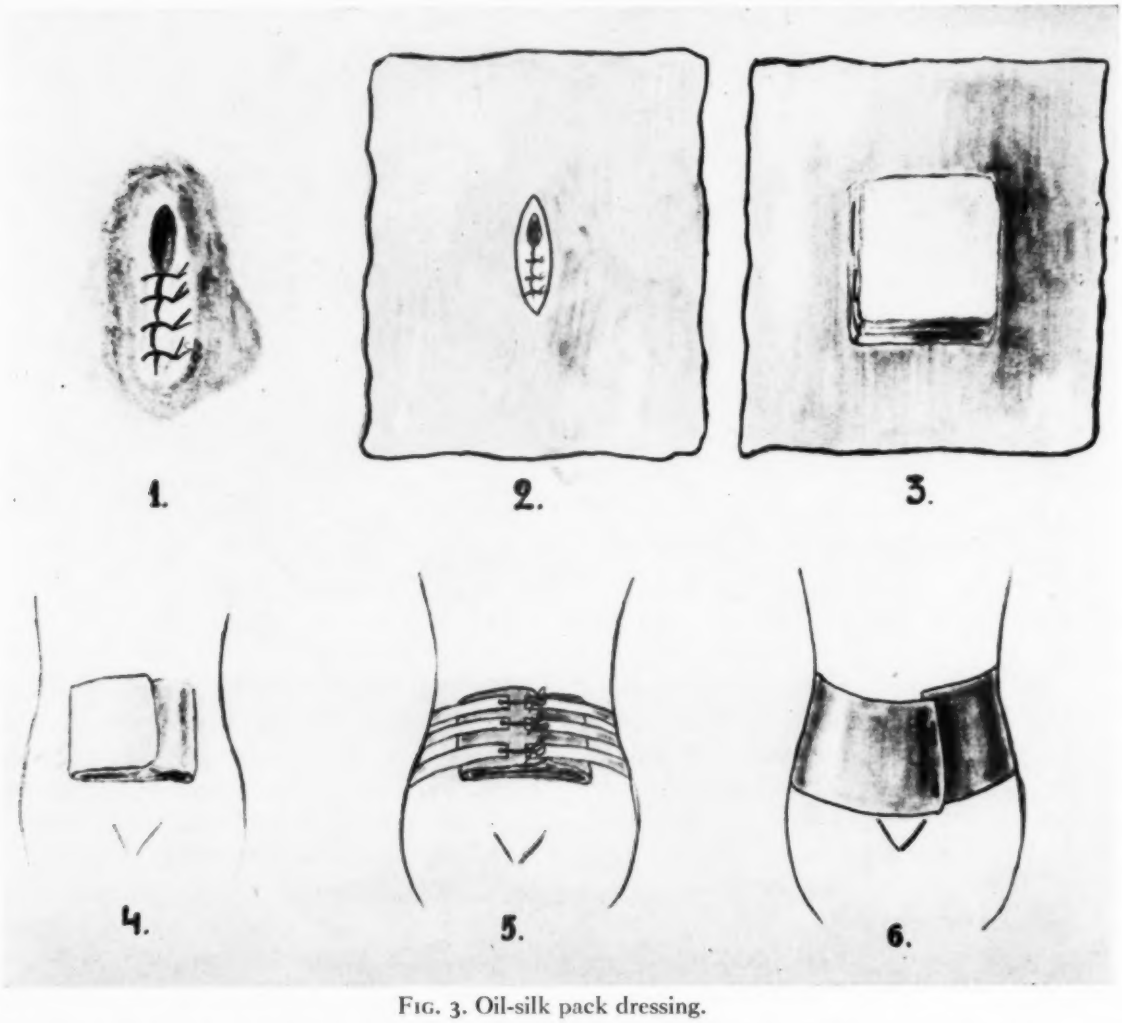


Fig. 3. Oil-silk pack dressing.

Complications Arising during the Drainage Period. During this period few if any complications are encountered. The patient is draining freely through the fistula and is cared for either by a suprapubic tube or by absorptive dressings like the oil-silk pack. Neither of these types of drainage is perfect. The drainage by tube is the easiest for the nursing staff but is, we believe, the cause of delayed union as compared with the free drainage into the oil-silk pack. (Fig. 3.) The latter dressing is very satisfactory if changed every two hours and twice at night; it gives the patient freedom of motion, removes the irritating tube and hastens the healing process.

We irrigate our cases daily beginning on the fifth day. The irrigation is done per urethra through a nozzle or through a urethral catheter gently passed until the cut-off muscle has been opened. We use

warm saline or mild antiseptics, washing until clear. In some instances the irrigation does more harm than good by initiating vesical spasms with hematuria leading to premature voiding with great pain and frequently with resulting hyperpyrexia and occasionally chills. Irrigation should then be discontinued and gentle washing of the bladder done by passing a small rubber catheter through the fistula.

During this period the patient is just as vulnerable to complications as any other surgical patient. It should be our endeavor to prevent them by anticipating them. Phlebitis is not uncommon, hypostatic pneumonia and flaring up of an old bronchiectasis are seen and in many instances can be prevented by sitting the patient up early and ordering breathing exercises hourly. We have seen cerebral hemorrhage, embolus, cachexia due to bilateral surgical empyema following atelectasis, massive necrosis of the inner side of both thighs following a faulty hypoclysis, heart failure, attacks of angina pectoris with sudden death, hemolytic icterus following transfusion, etc. All these complications are not specifically urological and should be cared for as in any other surgical case.

One must not omit the purely urological complication, namely, epididymitis. It does occur in about 30 per cent of the cases and is often the start of grave morbidity with severe toxemia and uremia. Vasectomy prevents this complication and should therefore be practiced routinely.

A complication not mentioned in the literature is "stump-vasitis." In some cases where vasectomy was performed as a prevention for epididymitis one notes about five to six days later a tender swelling at the site of the vasectomy. This tenderness may extend upward along the course of the vas and occasionally last seven to fourteen days with a low grade fever, toxemia and weakness. Reopening of the vasectomy wound may be indicated to establish drainage for the closed off vasoseminal tract.

Occasionally one sees a case of postoperative psychosis. Such cases usually die from physical and nervous exhaustion. However, many a decrepit old man has been kept alive and carried through to recovery by means of artificial feeding per nasal tube. Their obstinacy in refusing nourishment due to mental aberration, the strained protests sapping their strength and the loss of valuable time in waiting for improved mentality should be avoided by continuous nasal feedings via permanent tube with or without restraint of their hands. We saw a case of insomnia lasting four weeks which was completely cured on the day of his return home.

Another group of patients suffers from a mild toxemia with a mild septic temperature but very pronounced weakness with no evidence of an abscess. This state of affairs usually lasts eight to ten days and gradually subsides. We assume in many such cases the possibility of a low grade renal infection or more often ascribe it to a coexisting infection of prostatic bed, infected and closed off subtrigonal false passage or seminovesiculitis. In such cases we found the following procedure of value: Insertion of a small catheter into the bladder through the fistula and instillation of a solution one-third strength of hexylresorcinol every half hour by means of a bedside irrigator.

More serious are the cases with pyelonephritis and uremia. Most of them are fatal. Uremia per se is very rare; it usually is the accompanying mate of severe infection with abscess formation in the kidneys. The few cases that have recovered received continuous venoclysis with glucose, forced feeding per nasal tube and frequent transfusions. In addition to this, in every one of these cases competent drainage must be established and search for a closed off abscess cavity should be systematically carried out. Deep pressure in either lower abdominal quadrant will occasionally point to the site of perivesical suppuration and if suspected should be opened at once with a hemostat through the suprapubic wound.

Complications during the Closing Period. As the suprapubic fistula nears its complete

closure one again may expect complications. Delayed closure is the most important annoyance. This is not a natural phenomenon of a slow healing process but ally one may have a chill. This morbidity passes off in a few days and is usually relieved by reopening of the wound with a probe and insertion of a rubber dam.

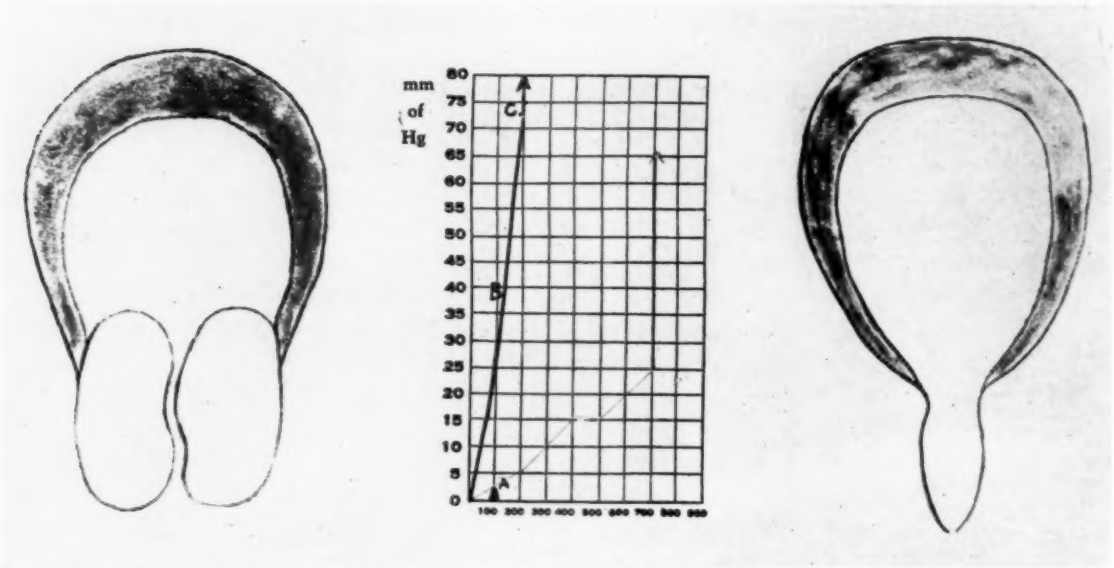


Fig. 4. Illustrating the cause of incontinence immediately after bladder closure which disappears in time. *Left:* Bladder wall before prostatectomy. *Right:* Bladder wall after prostatectomy. Middle chart illustrates the hypertonicity of the bladder wall.

is caused by a very definite pathological entity. It is not the broken down wound which will delay closure as one sees frequently bladder closure and competent urination with a widely gaping wound. The reason for delayed closure lies in the amount of tissue left behind in the commissure. While many such cases will close following forceful dilatations of the urethra with sounds or a Kollman dilator, a number of them will call for transurethral resection. It is wiser not to send the patient from the hospital with an open bladder as he invariably will seek another surgeon. The maximal time for closure of the suprapubic fistula should not exceed three to four weeks. If not healed by this time cystoscopic investigation will reveal the cause of the delay.

During this period one also encounters the phenomenon of "closing fever." In some cases the patient develops a temperature lasting two to three days and occasion-

The next troublesome complication to consider is incontinence. If one carefully analyzes the behavior of the bladder during this period, one sees a great many cases with partial or complete incontinence. This form of incontinence is very often not due to any injury to the sphincters whatsoever, but is the result of altered physiology of the bladder mechanism. As the prostate increases in size and begins to obstruct the vesical outlet, the bladder wall musculature or the detrusor becomes thickened and trabeculated. One sees obstructed bladders with the bladder wall measuring several centimeters in thickness in a vain effort to overcome the stenosis. While the obstructing factor is removed at operation the thick bladder wall remains with the resulting powerful contractions on evacuation. The initiation of the desire to void is easier, the responding reflex much quicker and the bladder contractions more powerful than in normal. A severe grade of

frequency and especially an intense urgency is established, thus simulating incontinence. Such incontinence will gradually disappear with the administration of sedatives. Very helpful is the wearing of a clamp in training the bladder to carry larger amounts of urine (Fig. 4). There is, however, a group of patients who remain incontinent permanently and must undergo some form of operation for its relief. This group of patients cannot ascribe their disability to faulty surgical technique only. One sees many cases showing no sign of incontinence after a very traumatic prostatectomy. We feel that the main cause of incontinence in this group is due to chronic infection of the prostate and periprostatic tissues with destruction of their elasticity, resulting in postoperative flabbiness and relaxation of the prostatic urethra. Here surgery is the only hope for cure.

Complications after Closing of Fistula. If the bladder neck is free from obstruction a suprapubic fistula once closed will not reopen. It will invariably reopen if partial obstruction persists. Prompt transurethral resection of the residual tissue should be done, not only to close the fistula but also to prevent reaccumulation of residual urine with or without stone formation. The urine after closure of the bladder will remain infected for a long period of time, in many instances pus is found for years without any subjective symptoms. This chronic mild grade of residual cystitis should only be treated when large quantities of pus are present otherwise it should be left untreated as no treatment can eradicate it.

In cases where soft phosphatic incrustations persist, we resort to irrigations with mild phosphoric acid solution (0.5 per cent) per catheter with satisfactory results.

COMMENT

The appreciation of the seriousness of postoperative infection, of the weakness and vulnerability due to loss of blood and the immediate institution of therapy at the first signs of impending complications will greatly reduce the morbidity and prevent mortality.

It would be good preventive medicine to establish the practice of follow-up examinations in every man who underwent suprapubic prostatectomy, not only to have him well but also to keep him well permanently.

If autopsy could be done routinely in every case there would be less diagnoses of pneumonia and more of atelectasis, less of cardiac failures with cachexia and more of pelvic abscesses, less of uremia and more of renal abscesses, less of acute pyelitis and more of faulty bladder drainage, etc.

MINOR ENLARGEMENT OF PROSTATE

EDWIN W. HIRSCH, M.D.

Attending, Urological Staff, Englewood Hospital

CHICAGO, ILLINOIS

MINOR enlargement of the prostate gland is a very common disorder encountered in middle aged and elderly men and presents a number of very important problems to the urologist. The first symptoms usually noticed are slight difficulty in starting the urinary stream, diminished voiding force, frequency of urination and the inability to terminate the voiding process in a rather abrupt manner. These symptoms may be caused by other conditions and do not necessarily bespeak for prostatic enlargement. We confirm the diagnosis of prostatic involvement by finding residual urine, rectal palpation of the gland and by cystoscopic examination. It is the aim of the urologist to relieve the patient's distress associated with urination, by a procedure which entails the smallest amount of inconvenience and danger to the patient.

Generally, the tendency has been to focus entire attention on the bladder neck, i.e., the area around and adjacent to the internal urethral orifice. Cystoscopic examination of these cases usually reveals an elevation of the lower lip of the bladder outlet which is the end result of proliferation of underlying prostatic gland tissue. This extension of the prostatic tissue into the bladder is regarded as a dam which by mechanical obstruction prevents the bladder from completely emptying its content. By removing the obstructing element, many urologists believe that the cause of urinary difficulty can be corrected. All too often however, removal of this tissue has not led to permanent correction of the urinary disorder. Let us therefore further study the underlying cause of minor urinary retention.

In evaluating the role which the prostate gland plays in the urinary process, we must not overlook the fact that the prostate is a muscular as well as a glandular organ. In several respects the prostate has functions which are similar to those of the urinary bladder. The prostatic urethra acts as a reservoir for the sexual secretions and expels them rapidly at the moments of ejaculation. During urination, the prostate works in unison with the bladder. The prostatic urethra dilates while the bladder contracts, and forcibly contracts after the urinary contents have been expelled. To this extent the evacuatory properties of the prostate resemble those of the urinary bladder.

Thus, it is to be seen that urinary difficulty and retention may result when any pathological process exists which interferes with the normal relaxation and contraction of the prostate. Minor enlargement of the prostate may be due to the abnormal retention of prostatic secretions. When this process has existed for a long period of time, the bladder muscle is caused to hypertrophy so that it can force the urine through a channel which will only partially open during urination. Prolonged retention also induces an irritation and ultimately an hypertrophy of the smooth and voluntary muscle at the outlet of the prostate, i.e., the cut-off muscle and the muscle adjacent to it. Such changes induce hypertrophy of the bladder muscle and it takes added strength for the bladder to force out the urinary content when the cut-off muscle is hypertrophied. Obviously then, some cases of urinary retention may be partially due to hypertrophy of the prostatic sphincter.

Another cause of urinary retention is the direct effect which the enlarged and swollen prostate exerts on the contractile power of the bladder muscle. I was led to

the adoption of this view as a result of my anatomic and histologic study of the bladder neck. Sections made of the entire bladder neck, show that the muscle of the bladder and prostate interlace and that inflammation of the prostate gland may extend into the muscular wall of the bladder. In such cases, the detrusors of the bladder will not be able to contract with their normal capacity and the prostate will not dilate as it normally should because of the inflammatory process within the muscular wall of the bladder. Thus it is to be seen that even though there exists a minor elevation or projection of the margin of the lower lip of the bladder neck into the lumen of the bladder, the fundamental difficulty may be due to the underlying prostatic tissue and that by removal of the apparently abnormal projection, we will not cure the condition which is interfering with the normal evacuation of the bladder.

Enlargement of the prostate gland in men past the age of 40 years begins in an insidious manner. Primarily, the process starts by a blockage of the ducts draining the minute prostatic glands. What is the cause of the secretion being retained within these tiny lobules? is the question which naturally presents itself. It may be due to infection of the normal prostatic secretion which changes the thin prostatic secretion into a thick inspissated substance which cannot flow easily out of the acini. Weakness of the muscles which surround the acini caused by inflammation and abnormal stretching may also be a factor. It is logical to assume that if the enveloping muscle of the prostatic lobules cannot contract forcibly, that the prostatic secretion will remain within the acini. Since the prostatic glands will continue to secrete, the lobules must enlarge to accommodate the increased secretion.

Some years ago, it was then believed that the obstruction to the outflow of prostatic secretion was not an important factor because in sections made of the prostatic urethra of men who died from prostatic disorder, it was seen that complete blockage of these ducts did not exist. However, considerable narrowing of these passages was seen. Faulty conclusions were drawn from these observations. Those who were eager to prove that mechanical blockage of prostatic secretion bore no relationship to prostatic enlargement, contended that the large prostatic ducts should be the structures first involved and therefore ought to be tightly closed by sclerotic changes. Subsequent research has shown that those workers who tried to discard the mechanical factor in prostatic hypertrophy entirely neglected focusing their attention on the site where the process began, i.e., in the tiny prostatic acini. Instead of looking within the prostate for the site of the initial process, those who wanted to disprove the mechanical block-

Fig. 1. Photomicrograph of the prostate gland of a dog showing cystic dilatation of acini caused by duct blockage. The microscopic appearance of the human prostate with duct blockage is strikingly similar. Cystic dilatation is one stage in the development of prostatic enlargement.

age theory, put all their attention on the prostatic ducts within the prostatic urethra.

Prostatic hyperplasia originates in one or several acini at any site in the gland. As the process continues, an entire prostatic lobule becomes plugged. All the accompanying processes such as hyperplasia of the glandular elements, hypertrophy of the prostatic muscle, the formation of prostatic nodules, the enlargement of the gland, develop as a result of the stimulus induced by infected stagnant prostatic acini.

I have stated that the underlying cause of the hyperplasia is an irritative factor plus stagnation of the prostatic secretion. The irritative factor is the toxic substance developed by bacterial growth within these acini. Many workers have admitted that signs of inflammation are present in most cases of enlargement but reasoned that such changes were accidental and resulted from infection produced by instrumentation, or were secondary to catheterization which brought about infection of the prostate. Though this conclusion seems rational, it is not supported by fact as any number of prostate glands show inflammatory changes though they have never been instrumented. In studying these glands, we see tissue changes which have existed for some time and are not the result of a recent infection. Besides, accidental infection would not be confined, as prostatic pathology is in many cases, to a single area at a considerable distance from the urethra.

The wide variety of pathological changes seen in prostatic enlargement, such as hypertrophy of the median lobe, hypertrophy of the lateral lobes, collar neck fibrosis, sclerosis of the prostate, commissural hypertrophy, are all based on the irritative factor induced by stagnant prostatic lobules. Hence correction of the disturbed function of the bladder cannot ensue unless the drainage from the prostate is facilitated. If this cannot be accomplished by non-operative measures then we are justified in using surgical measures.

SUMMARY

Minor enlargements of the prostate are the result of congestion, superimposed on the stagnation of prostatic secretion. The process is not confined to that portion of the prostate which projects into the bladder but concerns the prostatic glandular tissue, the smooth and striated muscle of the prostate and secondary connective tissue growth. If drainage of the prostatic ducts is promoted when the urinary retention is minimal, one can prevent the progression of prostatic hyperplasia.

ROENTGEN DIAGNOSIS OF DIAPHRAGMATIC HERNIA

ARTHUR S. UNGER, M.D. AND M. H. POPPEL, M.D.

Director of Department of Radiology, Sydenham and Gouverneur Hospitals, New York

Associate Roentgenologist, Gouverneur Hospital; Adjunct Roentgenologist, Sydenham Hospital; Assistant in Roentgenology, College of Medicine, New York University

NEW YORK CITY

INTRODUCTION

WE are presenting this subject because of the need for a detailed roentgenographic outline of differential diagnostic aids which may be of assistance in the early clinical diagnosis of the many cases of diaphragmatic hernia that we believe escape recognition in the ordinary routine Roentgen examination.

CLASSIFICATION

Diaphragmatic hernias may be divided into two types: congenital and acquired. Generally speaking, a diaphragmatic hernia is to be regarded as congenital until proved to be acquired.

The congenital type may be sub-divided into:

1. The form which is due to the complete absence of one half of the diaphragm. (Fig. 1.)

2. The form which is due to a partial defect in the musculature of one leaf of the diaphragm. (Fig. 2.)

3. The form which is due to a congenital enlargement of one or more of the diaphragmatic hiati.

The acquired type may be sub-divided into:

1. The form which is produced by a relaxation of the various hiatuses of the diaphragm. This type is usually secondary to an increased intra-abdominal pressure.

2. The form which is produced as a result of trauma.

There are two conditions which are so closely related to diaphragmatic hernia, especially in the differential diagnosis, that we have deemed it important to add them as a corollary in the classification. They are:

1. Thoracic stomach associated with a short esophagus. (Fig. 3.) This is not a true diaphragmatic hernia because the stomach has never been a part of the abdominal cavity, and has therefore never herniated. Because of the short esophagus, the stomach has developed in the thorax and must obviously remain there.

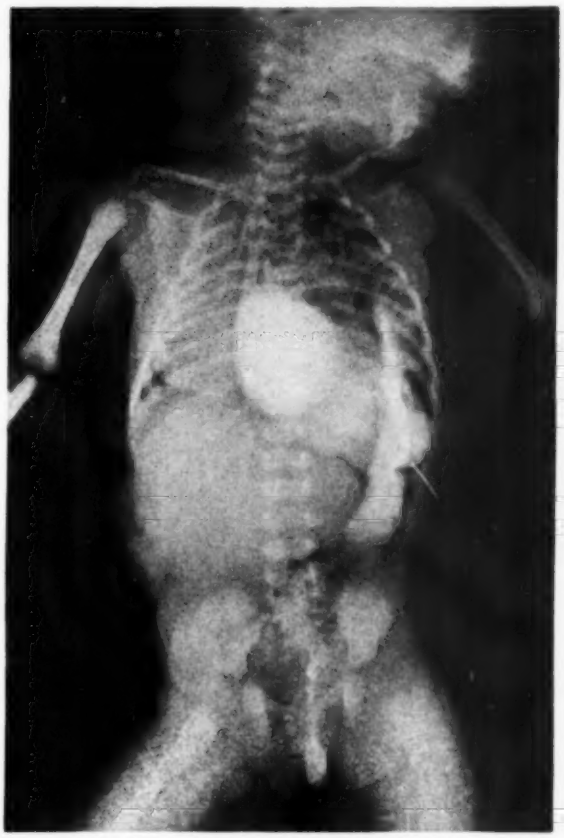


FIG. 1. Complete absence of the left diaphragm with colon and stomach in the chest.

2. Eventration of the diaphragm, with or without an associated megalo-colon. (Fig. 4.)

CLINICAL INVESTIGATION

We have carefully reviewed the symptoms and signs that all our cases presented before Roentgen investigation was instituted. From this study we have concluded that there is no clinical syndrome characteristic of diaphragmatic hernia. Nevertheless, there are certain symptoms and signs in the clinical picture which are suggestive of the diagnosis whenever they are present.

FIG. 2. Partial defect in the left diaphragm with the cardiac end of the stomach in the chest.

FIG. 3. Short curved esophagus associated with thoracic stomach. (*Courtesy Dr. Heiman.*)

We have found it suitable to make a composite listing of all the similar cases in our series and to present these suggestive symptoms and signs in the following ab-

FIG. 4. Eventration of the left diaphragm.

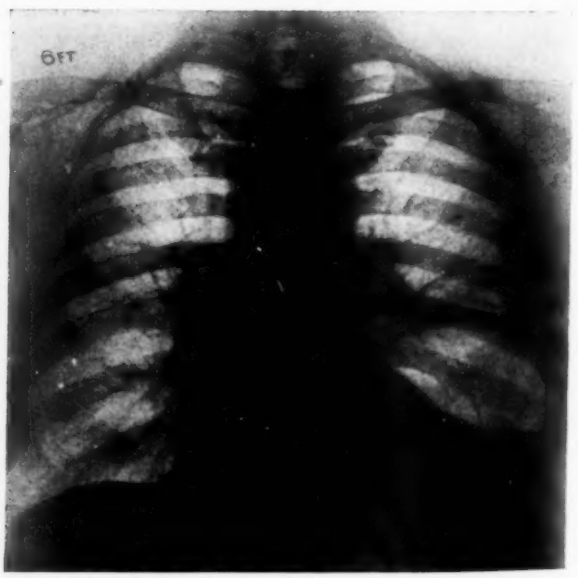


FIG. 5. Herniation of the cardiac end of the stomach into the left chest through a defect in the left diaphragm.

stract groups, in order to avoid a long discussion of the individual cases.

1. Any unusual marked epigastric dis-

Fig. 6. A large gastric herniation in a woman of 60.

comfort in a pregnant patient should stimulate search for an enlarged esophageal hiatus and for the herniating viscus.

2. Any gurgling, splashing, rumbling or other noises in the chest of a patient, should be investigated along these lines, especially if they are exaggerated in the knee-chest position.

3. Any delay in the onset of respiration in a new-born together with displacement of the heart, other respiratory difficulties and a persistent cyanosis might be due to a congenital hernia.

4. Any secondary anemia, associated with atypical, equivocal or evanescent physical signs in the base of the left chest should be investigated with the possibility that they may be due to a diaphragmatic hernia or one of the related conditions.

ROENTGEN INVESTIGATION. PART I

Roentgen Technique. Careful and thorough roentgenologic study will always

Fig. 7. Concentric shadows containing gas in the part of the left chest in a pregnant patient. Barium enema examination proved them to be colon.

enable the examiner to demonstrate diaphragmatic hernia or any of its related conditions, but on the other hand, it will invariably be overlooked unless it is actually searched for.

The Roentgen technique should include the following: preliminary fluoroscopy of the chest during inspiration and expiration. This will visualize the extent of the lung compression and the function therein. If a herniation is present we may see gas and fluid levels. This observation is especially important if the herniation is strangulated and no barium is able to enter the lumen of the involved portion.

There should be also included a complete fluoroscopic and Roentgen examination of the esophagus, stomach and small intestines, employing manual manipulation of the abdomen with or without compression. If negative, a barium enema examination of the colon must be made. It is sometimes necessary to have the patient cough or strain in order to demonstrate the lesion.

Careful posturing in the supine, Trendelenburg, lateral recumbent and lateral upright positions is very essential and should not be neglected in the routine study, as it is only in these positions that many of the herniations are demonstrated roentgenographically. The lateral views will show the antero-posterior distribution of both halves of the diaphragm, simplyfying the differentiation between hernia and eventration. This view is also valuable in demonstrating the absence of one leaf of the diaphragm. It must be borne in mind that the greater the degree of eventration the greater will the muscle of the diaphragm be thinned out, and consequently the more difficult to visualize. If a hernia is suspected and all the hollow abdominal organs are in their normal positions, it might be well to consider a solid viscus, such as the liver, spleen or kidney, to be the offender. If a flat abdominal film does not visualize these organs in their respective normal positions, it will be necessary to supplement the usual examination with either an intravenous injection of a small amount of thorotrast for the visualization of the liver and spleen, or with a retrograde pyelographic study using one of the urographic contrast media in a greater concentration than is ordinarily used, for the purpose of demonstrating whether the position of the kidney is normal or ectopic.

Fig. 8a. Defect in the left diaphragm through which the colon is herniating.

Fig. 8b. Same case as 8a. Appendix in the left chest.

ROENTGEN INVESTIGATION. PART II

Roentgen observations necessary for diagnosis.

The Roentgen examination must demonstrate the following if an early and accurate diagnosis is to be established:

Type and Site of Herniation. It may not always be possible to demonstrate the type or the site of the herniation. Whenever possible the observation should be included

in the report as it aids the surgeon in making his decision for the operative approach.

Contents of the Herniation. Almost all the abdominal organs except the rectum, urinary bladder and uterus have been found in the herniation.

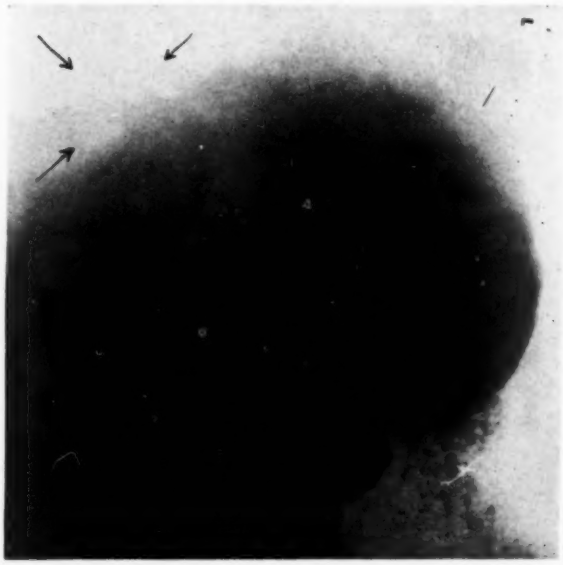


Fig. 9. Demonstration of gastric rugae above the left diaphragm.

Reducibility of the Herniation. It must be determined whether the herniation is fixed or whether it can be reduced. It may be possible to fill the herniation with barium if there is a strangulation at the neck of the herniation. Nevertheless, the diagnosis may be suggested by the concentric ectopic shadows containing gas and fluid levels situated above the diaphragm.

Length of the Esophagus. This observation is very important, as it explains why the stomach is in the thorax when the esophagus is short. This also enables the surgeon to avoid interference in the short esophagus type of case.

Size of the Esophageal Hiatus. It must be demonstrated whether the hiatus is normal or whether it is congenitally or otherwise enlarged. If the enlargement is acquired, it should be shown whether it is due to a pregnancy or to some other intra-abdominal increase in pressure. It is also important to know what passes the hiatus because in the short esophagus type of case associated with thoracic stomach, the stomach or duodenum may use this means of entering the abdomen.

Fig. 10. Herniation of the cardiac end of the stomach with a secondary proved malignancy in the herniated portion. There is a filling defect with distortion of the rugae. (*Courtesy Dr. Stone.*)

Diaphragms. We must study the structure, relative positions, shape, contour and movements of the diaphragms in order to determine their presence or absence, or the possibility of an eventration or other defect.

Costo-phrenic Spaces. We must determine the size, shape and illumination of these spaces, in order to rule out the presence therein of any displaced viscera.

Associated Pathology. It is necessary to demonstrate any associated pathology in addition to the presence of a diaphragmatic hernia or a related condition. The following conditions may be found associated:

1. Esophageal ulceration. The esophageal rugae are examined after the method of Berg as practised by Holmes and Schatski in this country. If the result is not satisfactory such as in the cases where there are shallow erosions instead of ulcerations, the examiner should seek the

FIG. 11A. Herniation through an enlarged esophageal hiatus.

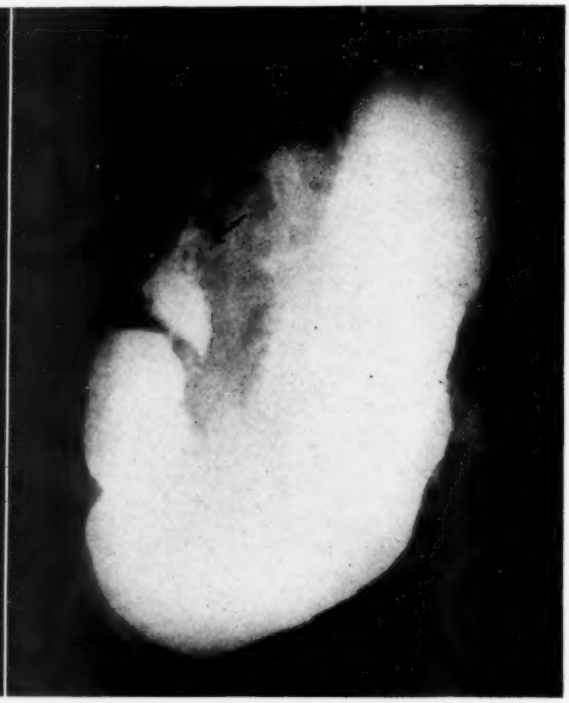


FIG. 11B. Same case as 11A. Duodenal ulcer seen in relief.

FIG. 12A. Large carcinoma in the sigmoid with large bowel obstruction.

FIG. 12B. Same case as 12A. Small bowel obstruction secondary to the sigmoid carcinoma.

aid of esophagoscopy. Manges and Clerf were able to observe eight cases in such a way.

Fig. 12c. Same case as 12a. Herniation of the cardiac end into the left chest as a result of the increased intra-abdominal pressure.

2. Gastric malignancy. We have encountered a case where the cardiac portion of the stomach which was herniated into the left chest, presented a large filling defect with distortion of the rugae, both characteristic of malignancy. Both conditions are visualized on the illustration. Later the findings were confirmed by operation. We believe that the association was merely a coincidence and not related to each other etiologically.

3. Duodenal ulcer. In this instance the ulcer was responsible for the symptoms and was consequently deserving of the treatment rather than the herniation which was the accidental finding.

4. Large bowel malignancy. We have encountered a case where the cardiac portion of the stomach was herniated into the left chest, through a defect in the left diaphragm as a result of a tremendous increase in the intra-abdominal pressure. This increased pressure was produced by a sigmoid malignancy which had perforated into a loop of ileum and as a result there had been produced both small and large

Fig. 13. Encysted pleural fluid resembling stomach in the chest.

bowel obstruction. In the roentgen routine of the gastro-intestinal examination the gastric herniation was found before the associated pathology in the small and large intestine.

5. Inflammation, as for example in the appendix. It is very important to know the position of the appendix on account of the possibility of the occurrence of an acute appendicitis. In one of our cases, the appendix was visualized in the left chest. This information will aid in accurate diagnosis and provide the surgeon with sufficient data to enable him to make a proper incision.

6. Secondary lung changes. The lung will be compressed and atelectatic in proportion to the size of the herniation. It is very important to know the extent of the lung damage, and to avoid any erroneous interpretation of this change.

Roentgen Investigation. Part III

Special Roentgen Features. The following Roentgen signs are especially sug-

gestive of the diagnosis and when present should stimulate further study.

1. The persistent absence of the stomach gas bubble (magenblase) is very suggestive of the presence of a diaphragmatic hernia, especially so if cardiospasm has been ruled out.

2. The presence of inspiratory elevation of one side of the diaphragm (paradoxical movement) is suggestive of an eventration of the diaphragm, especially so if there is also present a megalocolon.

3. The Roentgen visualization of any concentric shadows resembling stomach or intestine with or without gas or fluid levels, situated above the diaphragm, is suspicious of the diagnosis, especially so if the heart is displaced toward the opposite side.

4. Any apparent benign stenosis in the lower part of the esophagus should be investigated with the possibility of its being the esophago-gastric junction in a case of a congenitally short esophagus.

FIG. 14. A loop of colon between the right diaphragm and the upper border of the liver, resembling a diaphragmatic hernia clinically.

CONCLUSIONS

We have stressed the importance of the early recognition of diaphragmatic hernia, and have demonstrated that the Roentgen examination offers the only conclusive pre-operative proof of its existence.

It is hoped that the discussion herein presented will in the future enable the clinician and the roentgenologist to detect diaphragmatic hernia and its related conditions more readily than in the past, thereby providing a causal relationship to a group of variable symptoms and signs which have heretofore gone unexplained for long periods of time.

Accordingly, we would like to stress that there must be close cooperation between the clinician and the roentgenologist if accurate and early diagnoses are to be made.

REFERENCES

1. MANGES, W. F. and CLERF, L. H. Congenital Anomalies of the G.I. Tract. *Am. J. Roentgenol. & Radium Therapy.*, 33: 5; 650–668.

2. UNGER, ARTHUR S. and POPPEL, M. H. Diaphragmatic Hernia. *Am. J. Roentgenol. & Radium Therapy.*, 37: 4; 472–478.

3. UNGER, ARTHUR S. and SPEISER, M. D. Congenital Diaphragmatic Hernia. *Am. J. Roentgenol. & Radium Therapy.*, 15: 135–143.

A METHOD FOR REDUCING THE POST-OPERATIVE MORBIDITY OF CHOLECYSTECTOMY

THOMAS B. NOBLE, JR., M.D.

Visiting Surgeon Methodist, St. Vincent, and Hamilton County Hospitals; Consulting Staff Indianapolis City Hospital

INDIANAPOLIS, INDIANA

MUCH has been written about the operative mortality of cholecystectomy. This problem has been attacked from many angles by many men.[1] Intimately associated with mortality following gall-bladder surgery is morbidity, and we believe that this is an underemphasized feature of this surgery. Post-operative morbidity becomes the measure of the success of the surgeon in his dealing with the patient's diseased gall-bladder. It may develop as a result of operative technique and in so far as this is true may afford interest to the surgeon; or it may exist as a result of an incompletely treated pathologic process; this last pertaining to carcinoma or overlooked stones.

Any study of post-operative results may begin therefore with a study of operative technique. Surgery texts show that the incisions of Bevan,[2] Robson,[3] Kocher,[5] and others may be taken to represent established standards. More recently Devine,[4] Mortimer,[6] Mentzer,[8] Foster,[7] and others[9] have described ways and means of meeting the many problems that confront the surgeon during cholecystectomy, and suggest certain modifications of standard operations that are aimed at definite improvements in the degree of success achieved. To avoid too intensive study of modifications of related operative methods and the confusion resulting therefrom, we may understand that there are but three incisions for the approach to the gall-bladder, with many modifications of two of them.

First, there is the vertical incision through the abdominal wall, modified by and given the name of a number of men. Second, there is the subcostal incision, also modified and named as frequently. Third, there is the McBurney type incision which we have used to great advantage in cases of large hydrops, but which we believe to be impractical for routine use in other types of gall-bladder disease.

Since most operations are so closely related to others as to be mere modifications, the question arises quite properly—why bother with another? Critical analysis shows that the first incisions, namely, the vertical and subcostal, may sever any or all of the seventh, eighth, ninth, or tenth intercostal nerves which supply trophic innervation to the upper rectus of the same side. The fibers of the rectus sheath run transversely and at right angles to the path of a vertical incision. In early post-operative convalescence the normal elasticity of these fibers, plus the pull exerted by the oblique muscles and transversalis may cause separation along the deep suture line because of softening of catgut and lack of firm union which cannot be detected at the time, but which leaves an exposed raw surface where there should be smooth parietal peritoneum. The subcostal incision severs the muscle across its fibers and, when healing is ideal, leaves an intact abdominal wall by virtue of a fibrous union between the cut edges of the rectus, with a detectable gap between the severed ends of the muscle. If this incision is prolonged laterally, resulting hernia is common. Both vertical and subcostal incisions give open access to the area of the gall-bladder. This degree of exposure may necessitate the use of intra-abdominal gauze packs to restrain the various portions of the intestines from entering the field of operation. Respiratory movement during the operation may cause such gauze to abraid the peritoneum and to

offer traumatic insult to the celiac plexus at the same time. These facts alone, plus their rather obvious implications and

FIG. 1. Incision through anterior sheath of rectus, exposing muscle.

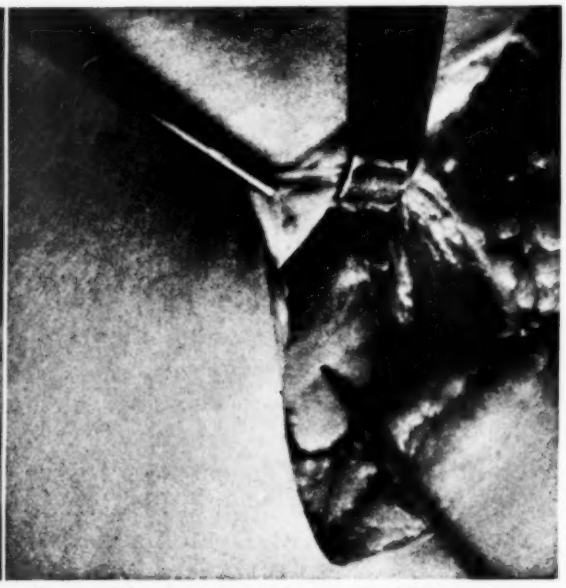


FIG. 2. Muscle retracted toward mid-line, exposing posterior sheath of rectus ready for perforation with small instrument.

sequelae, make it apparent why there are opportunities for the operative technique adopted by the surgeon to assume the role of causative factor in the production of post-operative morbidity. Three consequences of this fact may be mentioned that serve to prove the point that another surgical procedure for cholecystectomy is justifiable. If the nerves are severed subsequent atrophy of the rectus muscle and its sheath leaves a serious defect in the abdominal wall. The abrasion of peritoneum means adhesion, fixation, and lasting symptoms therefrom. Trauma to the celiac plexus means deeper anesthesia during operation and increased shock.

These, then, are some of the possibilities that may result from accepted surgical practice which make desirable, as well as justifiable, the development of an operation to eliminate any or all such possibilities attending cholecystectomy. Such an operation has been devised and used over a period of more than ten years, on all cases requiring cholecystectomy. In order to successfully accomplish its purpose of eliminating such undesirable attributes attending cholecystectomy, such an operation must not sever or split the fibers of the rectus muscle; it must do no structural damage to the sheath of the muscle; it must preserve all nerves; it must eliminate the need for mechanical retractors and gauze packs, and it must protect the liver from mechanical or thermal trauma due to too wide exposure. The operation must allow the patient an immediate return to physical activity without chance of hernia, it must favorably affect operative mortality, and must succeed in eliminating post-operative morbidity due to avoidable causes. Description follows.

The skin incision may parallel the rib margin one or two inches below that margin. The subcutaneous fat is pushed back a short distance from the sheath of the rectus. The fibers of the sheath are split parallel to their long axis, which is almost parallel to the rib margin. The upper or middle tendinous inscription may lie close to or below the most favorable region for entrance into the abdomen. This attachment of the muscle to the anterior sheath may be separated with the scalpel without

harm to the muscle or sheath. Then with the end of a Deaver retractor the muscle may be gently drawn toward the mid-line sion. The posterior sheath and peritoneum, and sometimes the continuation of the transversalis muscle, are penetrated with a

Fig. 3. Two diagnostic fingers of left hand filling opening through the posterior sheath.

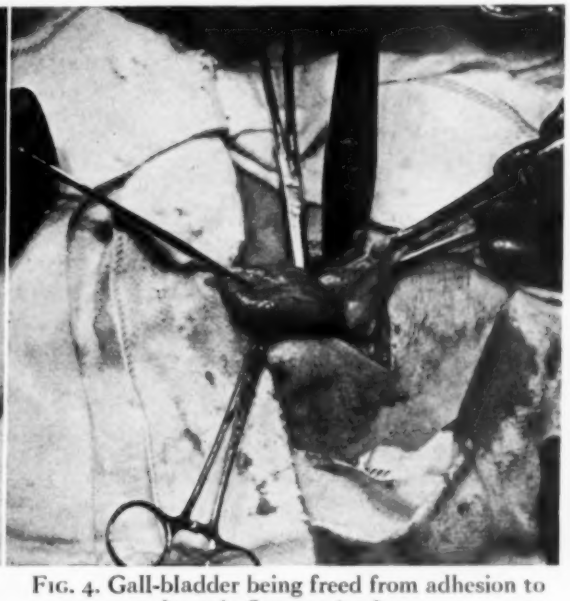


Fig. 4. Gall-bladder being freed from adhesion to hepatic flexure of colon.

Fig. 5. Gall-bladder ready for dissection from liver surface.

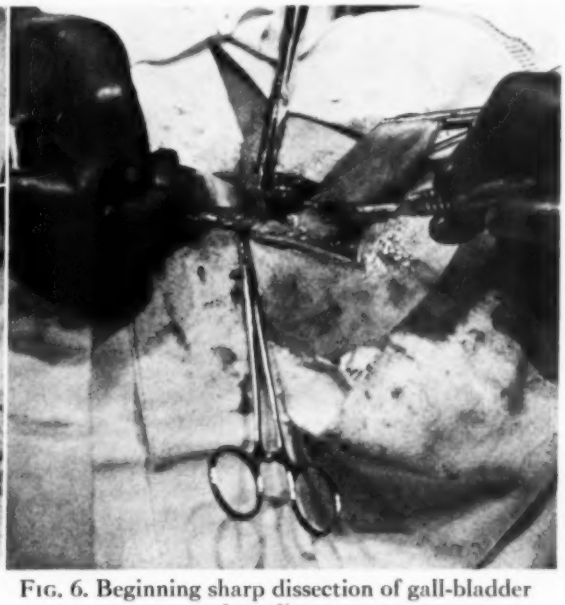


Fig. 6. Beginning sharp dissection of gall-bladder from liver.

without trauma to the muscle. This exposes to view the flat intercostal nerves lying loosely attached to the anterior surface of the posterior sheath of the rectus, on which they run almost to the mid-line before leaving it to enter the muscle. Lateral retraction of muscle may break nerves as with so-called paramedian inci- sharp pointed hemostat between two nerves, this method preserving the nerves and the sheath by splitting its fibers rather than by cutting across them. Palpation with one or more fingers introduced through the opening so made, gives determination of the extent and degree of the pathologic process to be studied and treated. We feel

that cholecystectomy is the procedure of choice except where there is carcinoma of the pancreas or an incarcerated stone in the common duct with some serious damage to the duct. The common duct is inspected, palpated, and probed except where there is present an acute inflammation with edema. A drainage tube, spirally cut, is anchored to the stump of the cystic duct by plain 2 catgut after completion of steps necessary to properly treat the condition found. The wound is closed with a continuous chromic 2 suture, bringing the tube out at the lateral margin or angle. The layers are closed in obvious order, and it is observed that they fall together much as do the layers during closure of a McBurney incision. The tendinous inscription is reestablished by the inclusion of the rectus in the suture closing the anterior sheath. The skin may be closed as one prefers. It is assumed that any

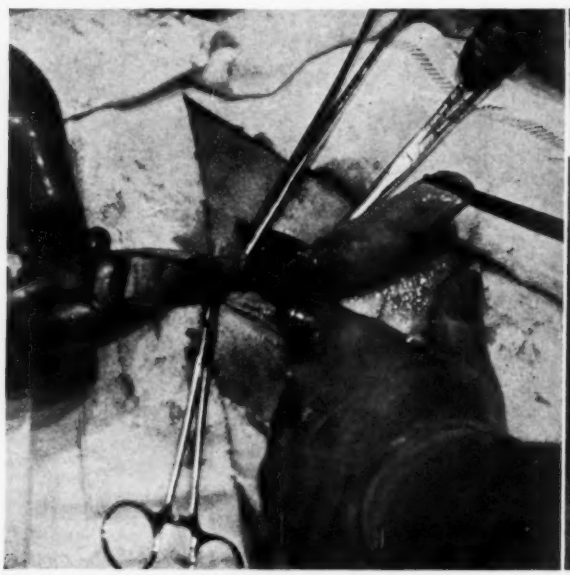


Fig. 7. Finger dissection exposing cystic artery.

Fig. 8. Ligature about cystic artery tied.

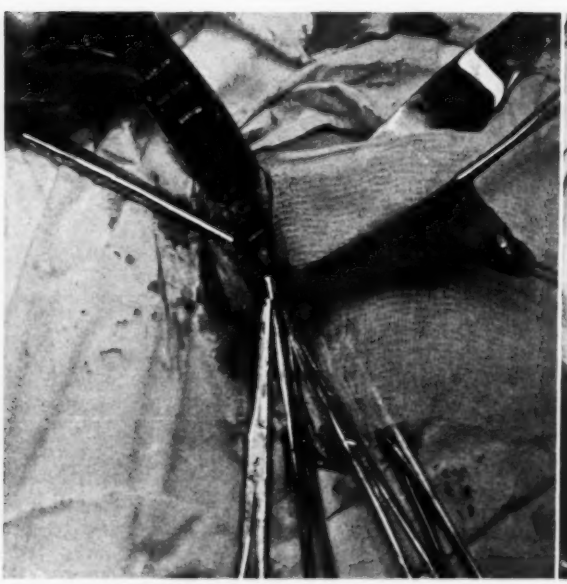


Fig. 9. Gall-bladder entirely freed from liver, completely extra-abdominal.

Fig. 10. Probe entering cystic duct.

measures needed to treat properly the pathologic process discovered after exposure and examination of the gall-bladder and surrounding area, will be continued as meets the particular choice of the surgeon operating. Since this has no bearing on the approach to the gall-bladder field, we may ignore this, other than to direct attention to the statistical study to follow.

It has been found that there are features favorable to the use of this method. A definitely lighter degree of anesthesia may be used throughout, particularly at the end of the operation and closure. The operative field is kept separate from the rest of the abdomen by the round ligament of the liver, the hepatic flexure of the colon, the omentum, and the parietal peritoneum. Since there is no bulging forth of the intestine through the wound, the mesenteric peritoneum may not be soiled, and danger of peritonitis to that extent eliminated.

Fig. 11. Ligature around cystic duct transfixing drainage tube.

Fig. 12. Tube in place, edges of posterior sheath ready for suturing.

Fig. 13. Suturing of posterior sheath completed.

Fig. 14. Suturing of anterior sheath including restoration of tendonous inscription.

Progressive dissection of the gall-bladder from the liver and any adherent structures limits most of the work of the operation to the smallest area directly in the abdominal wound. We have observed very satisfactory mortality statistics as will be given. There has been freedom from complaints such as those due to post-operative pylorospasm arising from fixation of the pylorus. Symptoms from immobilization of other viscera by adhesion have been absent. In the twelve years during which this method has been in constant and uninterrupted use there has been no hernia and no wound infection. This last accentuates the lack of motion that must exist between the edges of the sutured layers of the abdominal wall, following the use of this method of entering the abdomen, even though very early return to physical activity is believed to be a sound principle.

Against widespread adoption of this method may be listed a few definite features. The operative field is restricted as compared with either the vertical or subcostal. The ability to perform blind dissection with the fingers freeing the gall-bladder from any adhering structures must be exercised. Likewise, diagnosis of the extent and degree of the pathologic process by palpation alone is necessary. To one trained in the use of mechanical retractors and gauze packs, with wide free exposure for visualization of the gall-bladder area, this method will seem cramped, inadequate and hampering to complete surgery. Adequate exposure of the common duct may not seem possible at first. But the answer to these problems can be made best by a study of the results we have produced through the use of this operation. Our latest series of 194 cases taken in order consecutively runs as follows;

	Cases
Chronic Cholecystitis.....Total	129
No stones	24
Stones in the gall-bladder	70
Stones in the cystic duct	30
Stones in the common duct	5

There was no mortality. There has been no post-operative morbidity as measured by defect of or adhesion to the abdominal wall, overlooked stones, or complaint.

	Cases
Acute Cholecystitis.....Total	49
No stones	5
Stones in the gall-bladder	32
Stones in gall-bladder and cystic duct	55
Stones in the common duct with jaundice	7

Cholecystectomy was done in all these cases with results identical with those of the series of chronic cholecystitis with two exceptions. In two of these cases no probing of the common duct was made as is the choice plan where there is gangrene of the gall-bladder with marked swelling of the ducts. These two cases were reoperated later for pain caused by common duct stone and one recovered. The other case was operated elsewhere through another route.

Ruptured gall-bladder	9 cases
Cholecystectomies	6 recoveries without wound infection
Drainage and cholecystostomy	3 fatalities following drainage alone

This might seem to indicate that cholecystectomy were safer than cholecystostomy and drainage, but the truth is that three patients were too sick to survive, and six were not. Combined cholecystectomy, pylorectomy, and posterior gastroenterostomy; five cases, no mortality and desirable lack of morbidity. Combined cholecystectomy and nephrectomy for stones; one case with desired post-operative history. Angina pectoris complicated with acute gangrenous gall-bladder; one case fatality from coronary embolus on seventh day.

Any statistical study should include the time of legitimate return to ambulatory activity. These cases have been propped up in bed on the third and fourth day, and average cases reach the bedside chair by the seventh day. Return home has been by the tenth day, and at this time the patient has been able to care for himself. The psychic factor is most important here, for some have driven a car on the tenth day while others are yet frightened. There is an economic factor that may have been

suggested by the above figures. In this series of 194 cases it can be estimated conservatively that 200 weeks of hospitalization have been eliminated through the use of the described method. Accordingly it might be estimated that 200 months of working time have been saved. This is a guess, of course, but will bear scrutiny. A last point needs perhaps no mention. It is that there is a psychic as well as economic feature which helps determine the degree of post-operative morbidity. This has to be weighed carefully before a true gauge may be made of the presence or absence of post-operative morbidity.

CASE HISTORIES

The first case is cited in order to show that advantages gained by the use of this operation, if there be any, may be used to make operable cases which might otherwise be inoperable. Mr. O., sick seven days; age 52; temperature on admission 102; delirious; pulse 140; dehydrated with red cracked tongue; marked rigidity of the upper right abdomen; twenty hours pre-operative preparation with bicarbonate and glucose, fluids and ice, gave improved renal output but little else, and it became apparent that there was continuing leakage from the gall-bladder. Cholecystectomy of a gangrenous gall-bladder that had given rise to some considerable degree of local peritonitis was done. Convalescence caused some concern, but recovery was had without infection of the abdominal wound. His post-operative history has been completely satisfactory for more than a year now.

The second case is cited to show what is considered not unusual or illogical in regard to recovery from cholecystectomy in less serious cases. Mrs. B., not well for four years following an illness that we now know to have been a very marked degree of acute cholecystitis. Continual complaint and digestive disturbance during the four years. At cholecystectomy, done at the end of this time, the hepatic flexure of the colon and the duodenum were found to be so densely adherent over the small contracted gall-bladder, that sharp dissection with scissors was necessary to expose the gall-bladder. One large stone was in it, but the ducts were found small and empty to palpation, and examination with a probe into the duodenum through the common duct. Post-operative recovery was free of shock or the need for more than one-half grain of morphine total. Accordingly, the patient was out of bed on the fourth day and home on the seventh, able to care for herself. The later history has been desirable. No patient is forced for the purpose of making a show or record, and no patient is encouraged to be sicker than they should be. It is considered proper to take advantage of all possible gain, considered from the standpoint of some twelve years experience with this plan.

SUMMARY

1. Operative morbidity following cholecystectomy is worthy of serious consideration.

2. An operative procedure has been described which may eliminate some avoidable causes of post-operative morbidity, as well as improve operative mortality.

3. Statistics taken from the study of an unselected series of 194 consecutive cases are used to illustrate.

REFERENCES

1. Thorek, Max. Electrosurgical Obliteration of Gall-Bladder without Drainage. *Am. J. Surg.*, 32: 417 (June) 1936.
2. Bevan. Surgical Clinics of Chicago. Vol. 1, 1917.
3. Mayo. Keene's Surgery. Vol. 3. Philadelphia, W. B. Saunders Co., 1919.
4. Devine. *Surg. Gynec. & Obst.*, 44: 85, 1927.
5. Kocher. Surgery, 2: 535–536, New York. Macmillan Co., 1911.
6. Mortimer, E. Subserous Cholecystectomy. *Am. J. Surg.*, 6: 83 (January) 1929.
7. Foster, G. S. Technique of Cholecystectomy. *Am. J. Surg.*, 3: 144 (August) 1927.
8. Mentzer. *California & West. Med.*, 34: 345, 1931.
9. MacGuire, D. P. Subperitoneal Decortication in Gall-Bladder Disease. *Am. J. Surg.*, 32: 77 (April) 1936.

POST-OPERATIVE STUDY OF PEPTIC ULCER*

BENJAMIN M. BERNSTEIN, M.D., DANIEL DIAMOND, M.D. AND JOHN ZASLOW, M.D.

BROOKLYN, NEW YORK

THE last word has not been said in the controversy over medical versus surgical treatment of peptic ulcer. In evaluating the results, it is necessary to follow the statistical method in as large a series and for as long a period of time as possible. We offer, therefore, this study of seventy-one cases heard from by questionnaire, eighteen months to seven years following surgical intervention for the cure of peptic ulcer.

Before going into our discussion, may we quote some figures published by others for the purpose of both comparison and criticism. Hinton,[1] in a recent article, reviewed 114 cases and stated that 37 per cent were cured, 12 per cent were benefited and 51 per cent remained unimproved. In a review of 466 cases published in the *Proceedings of the Royal Society of Medicine* for 1934,[2] the authors divided their results into four grades and claimed satisfactory results in 76 per cent. Martin Friedman[3] reviewed 360 cases and claimed 34 per cent as being entirely well after ten years, adding another 41 per cent—those who have occasional pains—with only 25 per cent as failures. Demel, Gulcke and Haberer have also quoted figures which seem to be very high for successful results by surgery. Balfour[5] in 1935 presented a masterful study of more than 1000 cases with the claim of a large percentage of permanent cures.

To establish concrete criteria by which results may be measured is difficult. One writer includes patients who have occasional pains, in the "satisfactory" or "good" class; others put into this group patients who cannot eat all foods without having pain; still others attempt to estimate grades and degrees of "good" results. It would seem that the answer to only one question is of the utmost importance: have the patient's gastric symptoms been completely and permanently removed? If any other course is followed in order to estimate a percentage of cure, the variability of response creates naught but confusion and the conclusions are therefore unreliable. Any statistical study which includes complete, three-quarters, one-half, one-quarter and one-eighth cures for example, can be of no value.

In our series of seventy-one cases heard from, as already stated, eighteen months to seven years following surgery, there were sixty-two males and nine females. Most of them had duodenal ulcers. The average age at the time of operation was 42½ years, ranging from 12 to 72 years. The average duration of symptoms before surgery was four years, except in some cases of acute perforated duodenal ulcers.

AGE GROUPS

Below 20	2
20 to 30	9
30 to 40	19
40 to 50	21
50 to 60	13
Over 60	6

TYPE OF OPERATION PERFORMED

Gastroenterostomy	43
Repair and closure of operation	18
Sub-total gastrectomy	11
Pyloroplasty	4
Multiple procedures	6

PERCENTAGE OF RECURRENCE OF SYMPTOMS

	Cases	Per Cent
Gastroenterostomy	11	25
Sub-total gastrectomy	2	20
Repair and closure of operation	8	44
Pyloroplasty	2	50

Sixty-three per cent of all cases were well at least eighteen months after operation; 17 per cent for more than 5 years. We found

* From the Department of Gastro-Enterology, Medical Division, Jewish Hospital.

no relationship between the duration of symptoms before operation, and the percentage of recurrence. The same was true in regard to both sex and age of the patient.

We offer this series of cases without comment because the number is small, the operations varied, and the after care unknown, the latter factor being particularly the case in all such studies. There are some conclusions, however, which are worthwhile noting.

CONCLUSIONS

1. The criteria for results were on the *all* or *none* basis, that is, the question was: Was the patient perfectly well after having submitted to surgery?

2. In a series of seventy-one cases with varied types of surgical procedures 63 per cent were well at least eighteen months after operation.

3. There was no relationship between the duration of symptoms before operation and the likelihood of recurrence.

4. There was similarly no relationship between either age or sex of the patient and the percentage of recurrence.

5. The percentage of recurrence of symptoms was about the same following gastroenterostomy and sub-total gastrectomy.

REFERENCES

1. Hinton. *Am. J. Dig. Dis. & Nutrition*, 3: 59–62 (March) 1936.
2. *Proc. Royal Soc. Med.*, 27; 225–242, 1933–34.
3. Friedman, Martin. *Zentralbl. Chir.*, 62: 1456, 1935.
4. Demel, R. *Zentralbl. Chir.*, 62: 2185 (September 14) 1935.
5. Balfour, Donald C. *Ann. Surg.*, 102: 581, 1935.

ACUTE APPENDICITIS IN CHILDREN

FURMAN ANGEL, M.D., EDGAR ANGEL, M.D. AND ALEXANDER KIZINSKI, M.D.

Angel Hospital

FRANKLIN, NORTH CAROLINA

AMONG the numerous articles appearing on acute appendicitis in recent literature there have been many concerning this disease in infancy and childhood. The basis of this report is a review of the last 104 appendectomies performed on children under thirteen years of age in the Angel Hospital during the period from October, 1930 to October, 1936. During this same period there were 6282 surgical admissions of which 1097 were operated on for acute appendicitis. Children under 13 years therefore comprised 9 per cent of the total admitted for this condition.

TABLE I
AGE DISTRIBUTION IN YEARS

Age	3	4	5	6	7	8	9	10	11	12	13
Cases	3	3	7	8	3	6	9	13	20	14	18

As is shown in Table 1 the youngest patient was 3 years and there was an increasing incidence with increasing age. It is an uncommon disease in the very young. In 1917 Abt collected 80 cases under two years of age from the literature. A case in a child two days old has been reported by Corcoran, and numerous other cases in the very young have been seen, but certainly under two years of age it is a rarity.

TABLE II
DISTRIBUTION ACCORDING TO SEX

Author	Year	Cases	Females, Per Cent	Males, Per Cent
Alexander	1920	500	43	57
Beekman	1924	145	37	63
Beth	1908	100	48	52
Deaver	1910	500	37	63
Edberg	1926	754	48	52
McLanahan	1934	179	47	53
Seager	1926	61	33	67
Tasche	1931	111	54	46
Totals	....	2350	43	57

This series shows fifty-one females and fifty-three males which coincides with the usual reported incidence. Table II shows the sex distribution as reported by various authors and demonstrates a slight preponderance of males.

It is generally agreed that there is a definite seasonal variation, being more frequent in the summer and fall. This series showed a higher incidence in the summer months, over one-half of them occurring in May, June, July and August (Table III).

TABLE III
SEASONAL VARIATION

Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
8	6	7	4	12	14	15	15	10	3	3	7

The history may or may not be of value in the diagnosis. Symptoms are often exaggerated by the parents and may be misleading. Only 16 gave a history of a previous attack. Ninety per cent were admitted with pain as the initial symptom. Nausea and vomiting were practically always present. However, we have never taken the position that the triad of symptoms—pain, vomiting and fever—was necessary to make a diagnosis. The average temperature on admission was 99.3° in the acute unruptured, 99.8° in the gangrenous unruptured, and 100° in the ruptured with abscess or peritonitis group. Thirty-nine had received a cathartic or laxative for which two parties are to blame·

the indiscriminate use of cathartics by the family in the presence of abdominal pain, the prescribing by a physician without seeing the patient, and what seems to be even more inexcusable, the prescribing of cathartics by physicians in the presence of abdominal pain. Recently we saw a case where the family physician not only prescribed a cathartic but ordered it repeated every two hours. At operation, after five doses of magnesium sulphate, the appendix was found to be completely sloughed off. The average time elapsing before operation in the acute unruptured cases was 2.3 days, the gangrenous unruptured 2.6, and the ruptured, including both with abscess and peritonitis, 3.8 days.

We recommend operation in all cases except when a fatal outcome is obvious. We have always felt that an error on the side of radicalism was preferable to one on the side of conservatism since there is practically no danger to the ordinary appendix operation, the loss of time is slight, and a source of future trouble is removed. During the above period operation was advised in all cases except one when the patient was practically moribund on admission and death followed two hours later. The McBurney incision was used 102 times. It is time saving, affords a ready access to the cecum and appendix, avoids unnecessary manipulation, is practically bloodless, can be closed loosely without danger of evisceration, and in our experience has not been followed by acute intestinal obstruction as often as has been reported by those using a right rectus incision. Even in the presence of a diffuse peritonitis we believe that a short McBurney incision, carried out rapidly under gas or local anesthesia, to be the treatment of choice rather than the conservative treatment advised by some. Enterostomy was combined with appendectomy ten times and colostomy once, the latter where a ruptured appendix was associated with a strangulated sliding hernia. An appendectomy with drainage was carried out first; the hernia was next exposed and the adherent colon found to be gangrenous, and a catheter was sutured into the strangulated loop. A jejunostomy was then carried out through an upper left rectus incision. The hernia and colostomy were repaired two months later.

An associated ascariasis was found only three times, in one of which there was a mortality.

TABLE IV

SHOWING VARIOUS FACTORS INVOLVED IN SERIES

Type	Cases	Per Cent	Temperature on Admission	Number Receivnig Cathartic	Number Drained	Number having Previous Attacks	Shortest Time Elapsed before Operation (Hours)	Longest Time Elapsed before Operation (Days)	Average Time Elapsed before Operation (Days)	Deaths	Mortality Per Cent
Acute unruptured	61	58.6	99.3	14	2	13	½	7	2.3		
Gangrenous, unruptured	9	8.6	99.8	4	6	2	10	7	2.6		
Ruptured with abscess or peritonitis	34	32.7	100	21	33	1	24	8	3.8	5	14.7
Total	104	....	.....	39	41	16	....	..	...	5	4.7

Forty-one or thirty-nine per cent were drained, the method used being a Penrose to the appendix stump, a soft rubber tube to the pelvis, or both.

The postoperative treatment consisted in combating dehydration by frequent hypodermoclyses, the use of the Fowler position, small doses of codeine or morphine at regular intervals, nothing by mouth, and the use of small direct blood transfusions.

TABLE V
MORTALITY REPORTED BY VARIOUS AUTHORS

	Cases	Mortality, Per Cent
Abt	208	7.2
Farr	500	4.8
Hudson	100	9.0
Kirschenmann	18	5.8
McLanahan	179	3.9
Seeger	61	8.2
Tasche	82	8.4
Woodall	295	7.5

There were 5 deaths, a mortality of 4.7 per cent. Although attention has recently been called to the danger of pelvic appendicitis by Brunn[5] because of the fact that it produces atypical symptoms and the condition when finally recognized is likely to show a ruptured appendix, we still have a much more wholesome respect for the appendix which is subhepatic or at least runs along the lateral wall of the cecum and is directed toward the liver. The following cases demonstrate this condition.

CASE I. R. L. B., female, aged seven years was admitted April 18, 1932 with a history of having had cramping pains in the epigastrium and vomiting for one week. The family thought the child had worms and administered a saline purge. The pain persisted and twenty-four hours before admission a mass appeared in the lower right quadrant.

Examination revealed an acutely ill child, face flushed, temperature 101°, pulse rate 110, marked tenderness with rigidity of the entire right side, and a leucocyte count of 16,000. At operation, the appendix was found to be sloughed off at the base; there was some attempt at the formation of an abscess near the lower border of the liver. The perforation was repaired and a Penrose drain inserted toward the liver and the cavity packed with gauze. The wound was closed loosely around the drains with two silkworm gut sutures. Death followed twenty-four hours later from peritonitis.

CASE II. J. T., female, aged 10 years was admitted December 7, 1933 with a history of having severe generalized abdominal pain for forty-eight hours before admission which radiated to the right upper quadrant, associated with nausea and vomiting. She had received castor oil twice.

On examination she was found to be dehydrated, temperature 101 degrees, pulse rate 130 and respirations 24. The abdomen was distended and tenderness was generalized but no mass was palpable. The leucocyte count was 12,600 cells. At operation through a McBurney incision and under nitrous oxide and ether anesthesia a perforated subhepatic appendix with spreading peritonitis was found. The appendix was removed and a Penrose drain inserted to the stump and a soft rubber tube to the pelvis. One round worm was felt in the head of the cecum. Four days following operation the patient developed signs of obstruction. The abdomen was opened through a left rectus incision disclosing a generalized peritonitis. Her condition continued to become worse and she was given 200 c.c. of blood by direct transfusion. Death occurred eight days after admission from general peritonitis. Twelve round worms were vomited and three passed by rectum the day before death.

CASE III. G. P., female, aged 12 years was admitted July 8, 1931 after having suffered with severe abdominal pain in the right upper quadrant, and nausea and vomiting for eight days.

Examination revealed a markedly dehydrated patient, a weak pulse with a rate of 120, temperature 97°, skin moist and cold, and a leucocyte count of 6800 cells. There was general abdominal tenderness and rigidity, the latter more marked in the right lower quadrant. At operation through a McBurney incision which was lengthened upward, a gangrenous ruptured appendix, in contact with the surface of the liver, was found. There was a spreading peritonitis present. The appendix was removed and a Penrose drain inserted to the base of the liver and the appendix stump. Death followed

twenty-one hours postoperatively from peritonitis and shock.

The other two fatalities were also in those having a ruptured appendix with a spreading peritonitis. They are:

Case IV. L. G., male, aged 5 years was admitted March 5, 1932 complaining of pain in the epigastrium with nausea and vomiting, all of twenty-four hours duration. He had received repeated laxatives given by the family.

Examination showed a well developed but dehydrated male child, temperature 102°, pulse rate 130, generalized abdominal tenderness which was more marked over McBurney's point, and rigidity in the same region. The leucocyte count was 12,400 cells. Under ether anesthesia and through a short McBurney incision a perforated gangrenous appendix was removed. There was a large fecalith located near the center of the appendix. There was no attempt at localization of the process. A Penrose drain was inserted to the stump and a soft rubber drain to the pelvis. The usual closure was carried out. Both temperature and pulse rate remained elevated until his death three days later from spreading peritonitis.

Case V. G. L., male, aged 7 years was admitted December 13, 1930. Seven days previous to admission the patient had his first attack of abdominal pain. Shortly after this he became nauseated and vomited. Purgatives were administered by the family and two days later pain became so severe the youngster had to go to bed. He was given further purgatives. The day before admission he was seen to be acutely ill, the lips were cyanotic and parched, the right thigh flexed, marked rigidity of all abdominal muscles, marked generalized tenderness of abdomen, temperature 101°, pulse rate 120 and a leucocyte count of 11,600 cells. The diagnosis of acute ruptured appendicitis with generalized peritonitis was quite evident. At operation under ether anesthesia and through a McBurney incision, a gangrenous appendix ruptured at the tip, was removed. A general peritonitis was present. Penrose drains were inserted and the abdomen closed as usual. The postoperative course was stormy, the temperature staying in the region of 104° and the pulse rate near 160 until his death five days later from generalized peritonitis.

It is therefore seen from the reports and as shown in Table IV that there was no mortality in any case of acute unruptured appendicitis or in any case where there was a well defined abscess. Table V shows the mortality as reported by various authors.

CONCLUSIONS

One hundred and four cases of acute appendicitis under 13 years of age are reported.

There was a mortality of 4.7 per cent, all in cases with ruptured appendices with little or no attempt at localization.

Considering the fact that in children operation is usually delayed longer than in adults it would seem that the operative treatment is as satisfactory in one as in the other.

Operation was advised in all cases except one admitted in extremis.

The McBurney incision was used 102 times and a right rectus twice.

Tables showing sex distribution, age and seasonal incidence, mortality of various authors, and other factors involved are presented.

REFERENCES

Abt, I. A. Pediatrics. Phila., Saunders, 1924, vol. 3, p. 560.

Alexander, E. G. Appendicitis in children; a study based on 500 cases. *Penn. Med. Jour.*, 24: 135, 1920.

Beekman, F. Acute appendicitis in childhood. *Ann. Surg.*, 79: 538, 1924.

Beth, V. *Boston Med. and Surg. Jour.*, 49: 427, 1908.

Brunn, H. *Surg., Gynec. and Obst.*, 63: 538–592 (Nov.) 1936.

Corcoran, W. J. *Am. Jour. Dis. Child.*, 39: 277, 1930.

Deaver, H. C. *J. A. M. A.*, 55: 2198, 1910.

Edberg, E. *Acta chir. Scand.*, 60: 397, 1926.

Farr, C. E. Appendicitis in children. *Arch. Pediat.*, 46: 335–345 (June) 1929.

Hudson, H. W. *New England Jour. Med.*, 212: 670–672 (Apr. 11) 1935.

Kirschenmann, J. J. *Am. Jour. Surg.*, 16: 318–322, 1932.

McLanahan, S. *Am. Jour. Surg.*, 25: 14–18, 1934.

Seeger, S. J. *Surg., Gynec. and Obst.*, 42: 536–539, 1926.

Tasche, L. W. Appendicitis in children. *Am. Jour. Med. Sc.*, 182: 86–94 (July) 1931.

Woodall, C. M. A study of acute appendicitis in children under 12 years of age. *New York Jour. Med.*, 28: 322–325, 1928.

HYPERPARATHYROIDISM

DIAGNOSIS AND TREATMENT

J. E. JACOBS, M.D. AND J. DEWEY BISGARD, M.D.

OMAHA, NEBRASKA

CLINICAL SYMPTOMATOLOGY

THE history, etiology and clinical pathology of hyperparathyroidism have been reviewed previously.

The disease may occur at any age, appearing usually between the ages of 35 and 55 years, and is more than twice as common in women as in men. The youngest case on record was 13 years old and the oldest was a woman of 69 years (Hunter and Turnbull).

The symptomatology can be divided into three groups:

1. That due to the hypercalcemia per se,
2. That related to the skeletal changes, and
3. That related to the increased excretion of calcium and phosophrus in the urine.

1. *Hypercalcemia.* Hypercalcemia decreases the excitability of the nerve muscle apparatus, the antithesis of tetany, the symptom complex produced by hypocalcemia. A greater electrical stimulus is required to cause contraction of muscles than in the normal and there results hypotonia, lassitude, ptosis, constipation, flat feet, vasospastic phenomena, and symptoms of weakness of the cardiac musculature. Insomnia, hypochondriacal states and neurasthenia occur. Often these symptoms are so vague that their existence becomes obvious only after they have disappeared. Pemberton, John and Geddie present a few cases with mainly gastrointestinal symptoms, such as vague pains or attacks of acute abdominal pain, vomiting, hyperacidity, constipation and anorexia. Taylor, Weld and Sykes showed that marked vascular changes occurred in the gastrointestinal tract of dogs and concluded that the effect was neurogenic and not due to the hypercalcemia because the effects were mitigated by full atropinization of the dogs.

In many cases the muscular weakness is outstanding. In some, it is so profound that the movements resemble those of cases with pseudohypertrophic dystrophia. Frequent falls, pain in the back and extremities and absent or diminished reflexes are common features which may result from the weakness alone. Four methods have been used for demonstrating muscular hypotonia.

1. The determination of the milliamperes needed to provoke muscular contration (Oppel).

2. The finer method of determining chronaxy, that is, bringing in the time factor of the muscular contraction, has been of use.

3. Moving picture will show the slow and weak action of the muscles, and allow comparison preoperatively and postoperatively (Rowntree).

4. The electrocardiogram shows that the muscular weakness does not spare even the heart muscle, this being proven by shortening of the R-T interval.

In a few cases the muscular atony is accompanied by coldness and bluish discoloration of the extremities and this has been such a prominent feature as to lead to a diagnosis of Raynaud's disease. Pressure on cranial nerves by the softened and deformed skull, or on the spinal root or cord in cases with collapsed vertebrae has led to diagnoses of cerebral affections or spinal cord tumor. All of the group manifestations mentioned have been designated "formes frustes" of hyperparathyroidism.

2. *Symptoms Referable to Skeletal Changes.* These vary in severity from

cases showing absolutely no symptoms referable to bone to ones in which the skeleton becomes practically non-existent and which, in fact, may end fatally from sheer inability to raise the thorax in respiration.

It would appear from experimental studies that there must exist a stage in the process of decalcification when osteoporosis alone exists, that is, before the added features of cysts, tumors and fractures have appeared. Thus in mild forms this disease theoretically may remain at this stage indefinitely and never develop true von Recklinghausen's disease. They may go unrecognized and be treated for flat feet, arthritis, neuritis, neurasthenia or ptosis.

In a pronounced case of hyperparathyroidism the patient drags his weak legs along slowly, a gait so characteristic that Rowntree has stressed its importance. Pain in the back and in the arms and legs is a common and often early symptom. It may be dull, deep and boring, or so severe that it requires the frequent use of narcotics. It is often made worse with exercise and is associated with stiffness of the joints so that a diagnosis of arthritis, neuritis or rheumatism is often considered.

The bones are often tender to pressure and this, as also the pain, may be localized to one bone, or may be generalized. Both tend to become more widespread as the disease progresses.

The incidence of pathologic fractures is probably more or less proportional to the degree and extent of skeletal decalcification. Not infrequently such fractures have led the way to an investigation by which the disease has been recognized. In advanced cases there may be innumerable fractures involving most of the bones of the body with collapse of vertebral bodies, of the thoracic cage, and of the pelvis. From the malunions and non-unions which often occur, there results more or less complete disability (to bedfast existence) and deformities.

The bones often bend without fracturing and this superimposed upon the ununited and malunited fractures occasionally results in such grotesque deformities that the victim is scarcely recognizable as a human being. The head enlarges either symmetrically or asymmetrically, and the spine shortens and bends into a kyphosis or scoliosis as the vertebrae collapse. There is often an appreciable reduction in height.

Tumors, due to underlying cysts, may be an early manifestation of the disease. It is especially apt to occur in the jaw and be treated as an epulis for years before the underlying condition is recognized (Albright, Aub and Bauer). Spontaneous fractures often occur through these cystic areas.

As has been stated previously, there is an abundance of evidence to indicate that there are two dicta which must be satisfied before a diagnosis of hyperparathyroidism can be made, namely a negative calcium balance and some involvement of the entire skeleton in the process of decalcification. Ballin and others have proposed that hyperfunction of the parathyroid glands is responsible for a variety of lesions of bone characterized merely by decalcification or osteoclasis of the entire or of merely a small portion of the skeleton such as giant cell tumors, osteitis fibrosa cystica localisata, Paget's disease, ankylosing arthritis, etc. Because these conditions occur in the absence of one or both of the essential criteria of hyperparathyroidism, the proposed relationship has not attracted much support.

Although the decalcification of hyperparathyroidism involves the entire skeleton, it may become more pronounced in certain portions or give rise to complications with symptoms referable principally to a limited area. Thus the decalcified vertebrae under weight bearing may collapse or develop pathological fractures, and give rise to advanced deformity and symptoms before involvement of other portions of the skeleton becomes manifest.

Regarding arthritic manifestations of hyperparathyroidism Funsten states: "The occurrence of arthritis is frequently responsible for the first symptoms of parathyroid disease. It may be recognized by symptoms of paroxysmal pain in the spine and abdomen, deformity of the spine, other joint involvements with secondary ankylosis." This brings up the question whether ankylosing polyarthritis has any relationship to parathyroid function. Oppel, the proponent of this theory and his followers, have failed to demonstrate the presence of a negative calcium balance in these cases. Further discussion follows under Differential Diagnosis.

3. Symptoms related to the increased excretion of calcium and phosphorus in the urine, (four or more times the normal amount) are polyuria, and, necessarily, polydipsia and those resulting from calculi and impaired renal function. Albright, Baird, Cope and Blomberg found an incidence of 52 per cent of renal stones, as evidenced by roentgenograms and postmortem examination, in a series of eighty four active cases of hyperparathyroidism reported in the literature.

Since many of these cases had received treatment of the renal lesions months or years before, the skeletal changes and other manifestations of the disease had become sufficiently advanced to lead to recognition of the fundamental disorder, these investigators did routine blood calcium and phosphorus determinations upon all patients with renal calculi observed in the urological department of the Massachusetts General Hospital. A diagnosis of hyperparathyroidism, previously unsuspected, was made in four of the cases studied. Two were operated upon and in both of these parathyroid adenomata were found and removed.

The renal lesions may be classified into three types, the division depending principally upon the duration and severity of the hyperparathyroidism.

Type I. Calcium phosphate stones in the renal pelves, with or without pyelonephritis.

Type II. A condition which simulates both chronic glomerular and vascular nephritis. It differs from Type I in that the deposits are in the renal parenchyma rather than in the pelves, and from Type III in that the kidney shows long standing changes and may be the only organ in which deposition of calcium has occurred.

Type III. Acute renal failure with anuria and death in a few days, with calcium deposits in the renal parenchyma as well as in other organs, but with no other chronic renal changes. This may develop rapidly and cause death before skeletal changes have progressed very far.

Albright cites cases that have been erroneously diagnosed as diabetes insipidus because of the symptoms of polydipsia and polyuria. Renal colic and hematuria or persistent albuminuria may be the only presenting symptom of hyperparathyroidism; in fact, Albright and Bauer believe that hyperparathyroidism is a fairly common cause of urinary stone and that as cases with stone are investigated more commonly from this point of view hyperparathyroidism will be recognized more often and at an earlier stage of the disease.

DIAGNOSIS

The diagnosis of hyperparathyroidism, although suggested by the symptoms, depends upon confirmatory laboratory data and roentgenograms.

The disease has a greatly varied symptomatology. It must be considered in patients presenting various skeletal lesions, cysts, tumors, and fractures. It embraces most of the specialties of medicine. The dentist or oral surgeon must be mindful that an epulis or cyst of the jaw may be only a local manifestation of this generalized disease. The pain may be generalized and severe or limited merely to the low back or may suggest foot strain. The symptoms may simulate those of neurasthenia and of various gastrointestinal and renal diseases. Thus Albright states that "one must think of the diagnosis very often, but be prepared to be disappointed almost, but not

quite, equally often. The rare ten-strike in the borderline case makes the effort worth while."

Fortunately, hyperparathyroidism is one of the diseases that may be diagnosed by the laboratory per se. Repeated examinations almost invariably will disclose one or all of the characteristic findings of hypercalcemia, hypophospatemia, hypercalcenuria and hyperphosphaturia.

The history of the development of the association of these laboratory findings has been given.

It has been shown repeatedly that the normal range of the blood calcium is 9 to 11 mgms. per cent of blood serum, and that of the inorganic blood phosphorus ranges from 3 to 4 mg. per cent. The plasma contains phosphatase in quantities from 2 to 4 units, method of Bodansky. Since this enzyme is present in large quantities when and where calcification is taking place, quantitative determinations of it may prove, with the accumulation of more data, a valuable diagnostic aid and an index to the activity of the disease. Phosphatase is present in unusually large quantities when bone is disorganized, according to Kay.

Albright, Aub and Bauer state that "a serum phosphorus below 3.5 mgms. per cent and a calcium above 11 mg. per cent should be regarded with suspicion (the serum taken fasting)." Snapper reported a case of hyperparathyroidism with a serum calcium as high as 23.6 mgms. per cent and Hannon, Shorr, McClellan and DuBois reported a serum phosphorus as low as 1.4 mgms. per cent. It must be remembered that renal insufficiency may raise the level of serum phosphorus.

Under certain biological conditions, hypercalcemia may occur as a normal phenomenon. Biddle and Reinhart have shown that the serum calcium of certain birds increases very considerably at the period of ovulation and Hess and Bills state that this also occurs in the female cod in the spawning season. Hunter and Aub have reported the production of severe hypercalcemia (19.8 mgms. per cent) in a man by overdosage with parathormone and that a marked rise of the non-protein nitrogen of the blood follows the administration of large doses of parathormone. It appears that in the long standing cases there is increased absorption of calcium and phosphorus from the gastrointestinal tract and that excretion of calcium into the gastrointestinal tract is not dependent on the level of the serum calcium (Albright, Bauer, Claflin and Cockrill). Richardson, Aub and Bauer found that the urinary output of calcium and phosphorus on a low calcium diet was six to seven times greater than was found in normal individuals under identical conditions, and that these changes were equivalent to those of a normal individual receiving one hundred units of parathormone daily.

Bence-Jones proteinuria can occur in von Recklinghausen's disease. Albright reported three cases in 1935, in which it disappeared after operation, but not immediately, and they assumed that the excretion of this substance in hyperparathyroidism can occur only when there is associated skeletal changes.

The roentgen findings are variable. Although the skeletal changes may be so slight as to be almost imperceptible there is invariably increased radiability, and in advanced cases, deformities, cysts, tumors and fractures; the first being fundamental, the others secondary changes. As a metabolic disease hyperparathyroidism must exert this fundamental action, demineralization, of the entire skeleton. However, the changes may be so much more pronounced in certain areas as to obscure the generalized nature of the disease and to suggest the occurrence merely of a localized lesion. Some authors consider the type of decalcification pathognomonic of the disease, consisting, as it does, of a uniform, miliary, granular mottling, best observed in the calvaria, thinning of the cortex and trabecula of the spongiosa and the presence of areas of subperiosteal resorption in the long bones and phalanges. Therefore, in

doubtful cases, it is essential to decide at once whether one is dealing with a generalized or a localized disease. Furthermore, a disease may be polyostotic without being generalized. It must be emphasized that at first sight the secondary bone changes, as cysts, tumors and fractures, may make one think that the skeleton is involved in a spotty manner, but a more detailed study will reveal that the decalcification is uniform and generalized. Of course, immobilization due to a fracture may enhance the decalcification in the involved part. Absence of the lamina in the tooth sockets seen in *x*-ray pictures has been emphasized. Deformities demonstrable roentgenographically may be very extensive and obvious. Lateral roentgenograms of the lumbar vertebrae often disclose biconcave discs (fishbone vertebrae), which occur because the softened vertebrae can no longer withstand the tendency of the nucleus pulposus of the intervertebral disk to expand. They are not pathognomonic of hyperparathyroidism but may occur in such diseases as osteomalacia, multiple myeloma, etc.

The cysts, if present, are usually multiple, but need not be. A cortical cyst is especially suggestive of this disease. The tumors or osteoclastomas usually occur in the jaws, at the ends of the long bones, or in the ribs. In contradistinction to the cysts, they occur only where there is cancellous bone. Some cases show peculiar lesions in the terminal phalanges, in that the edges of the bone everywhere are fenestrated, suggesting a complete lack of cortex. Fractures, when present, usually occur through cysts or tumors.

Roentgenographic examination of a large part, or of the entire skeleton will often aid materially in establishing the diagnosis.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis will be discussed under three headings representing the three systems which are involved, namely, A. the skeletal system, B. the muscular system, and C. the renal system.

A. Early in the course of the disease or in the mild and slowly progressing forms, the diagnosis may be unsuspected for a long period of time and the patient be treated for rheumatism, sciatica, lumbago, sacroiliac strain, etc. The mere demonstration of demineralization of the skeleton does not make the diagnosis but leads merely to a suspicion and can be established only by sufficient laboratory evidence to indicate the existence of a negative calcium balance. There are several other maladies in which there occur similar or identical skeletal changes, some occurring as localized lesions, others involving the skeleton as a whole.

1. Physiological osteoporosis.

Bone becomes demineralized simply as a normal physiological process under certain circumstances, and is characterized by qualitative normality except for a quantitative reduction of its mineral content. Osteoporosis is a normal accompaniment of senescence and of disuse atrophy. It may become rather extreme in debilitated bedfast patients.

A generalized osteoporosis has been observed in cases of exophthalmic goiter of very long standing and the condition may persist for years after the thyrotoxicosis is relieved.

Prolonged calcium starvation robs the bones of their lime salts and this results either from a diet grossly inadequate in calcium content or from circumstances which interfere with its assimilation from the intestinal tract. Calcium is excreted before it can be absorbed in cases with external jejunal fistulas, with gastrocolic or jejunocolic fistulas and with severe prolonged diarrhea from any cause. In certain vitamin deficiency diseases (discussed later) in pancreatogenic steatorrhea and in idiopathic steatorrhea (non-tropical sprue) there is failure of assimilation presumably because the calcium or the fat soluble vitamins are held in an unabsorbable combination alone or with the fats.

As in hyperparathyroidism, the roentgenograms show increased radiability of

the bones. Fractures and deformities may occur, and there may be pain in the bones. In the senile form the serum calcium is normal, the serum phosphorus is reduced (often about 3 mgms. per cent) or normal, the plasma phosphatase and the urinary excretion of both calcium and phosphorus are normal. Biopsy specimens of bone fail to show fibrosis of the marrow and an increase in osteoclasts. Senile osteoporosis is perhaps the most difficult to differentiate because the resemblance to mild cases of hyperparathyroidism. As stated previously, generalized osteoporosis without fibrosis has been observed in experimental hyperparathyroidism by Jaffee et al. They showed that when the decalcification was less severe, generalized thinning of the bones without marrow fibrosis occurred (osteoporosis) but when more rapid, generalized decalcification and secondary marrow fibrosis resulted (osteitis fibrosa). Thus Moreau drew his conclusion that in all conditions showing decalcification and disturbed calcium metabolism there was an associated hyperactivity of the parathyroid glands.

Osteoporosis resulting from calcium starvation can be differentiated by the presence of a hypocalcinemia and a hypocalcinuria.

2. Osteitis Deformans (Paget's disease).

In the opinion of most authorities osteitis fibrosa cystica (von Recklinghausen's disease) and osteitis deformans (Paget's disease) are two distinct clinical entities. However, pathologically and clinically, they have so many features in common that some question as to a common etiology has been raised.

Ballin states,

> Now the results of parathyroidectomy in Paget's disease confirm the opinion that the two diseases are identical and can be controlled by parathyroidectomy. The French, who have given a good deal of consideration to the question of Paget's disease, have come to the conclusion that one should explore the parathyroid region to ascertain as to whether the case is a von Recklinghausen's disease or a Paget's disease.

Albright, Aub and Bauer, although disagreeing with Ballin and Morse in stating that the signs and symptoms of the two diseases are not the same, include in their classification of "clinical types" of hyperparathyroidism those simulating or complicated by Paget's disease in which findings characteristic of both diseases are present. Points of difference which seem to preclude a common etiology are that (a) although Paget's disease is often polyostotic it is never generalized, which is almost inconceivable for a metabolic disease; (b) the metabolic changes are not those of hyperparathyroidism, i.e., the serum calcium is normal or only slightly elevated (11 mgms. per cent) and the serum phosphorus is normal or slightly elevated, and the plasma phosphatase, given a comparable degree of disease of bone, is considerably higher than that observed in osteitis fibrosa cystica (Kay), also the calcinuria and phosphaturia are usually not present when the patients are run on a test diet; (c) the roentgenograms show spotty distribution of the lesions and the presence of normal bone somewhere in the body, especially the small bones of the hands. The involved bones, often the long bones, in most instances show characteristic enlargement (hyperostosis), are often soft and bowed, and show a thickened cortex in both *x*-ray and macroscopic examinations. The skull is usually thick and has a moth-eaten appearance radiographically. The weight bearing bones, including the sacrum, are often involved, and the apposition of new subperiosteal bone preponderates over resorption; (d) malignant bone tumors develop in a fair number of the cases of Paget's disease, such as is rarely, if ever, observed in hyperparathyroidism; (e) fractures (frequently spontaneous) occur very often in hyperparathyroidism and seldom in Paget's disease; (f) renal calculi are the exception in Paget's disease but are found very often in hyperparathyroidism. It is true that histologic sections of bone from these two diseases may be similar and therefore it may be difficult

to make a diagnosis on this basis alone, but in general the course pattern of the trabeculation of the affected bones, which in itself is almost pathognomonic of Paget's disease, contrasts with the sparse irregular trabecula of osteitis fibrosa cystica.

Bauer comments that Paget's disease occurs in older people, usually males, and is regularly associated with marked arteriosclerosis, whereas hyperparathyroidism is met in all age groups, more commonly in females, and arteriosclerosis is not a common finding. The late Prof. Schmorl in an exhaustive pathological study of the largest group of cases of Paget's disease ever reported, (one hundred and thirty-eight personally observed autopsies and seven other cases) was unable to demonstrate the presence of a parathyroid tumor or of abnormal histologic changes in these glands, and thus found no evidence to indicate that Paget's disease was due to hyperparathyroidism.

Directly opposed to the theory of hyperfunction in regard to the etiology of Paget's disease is that of hypoparathyroidism. In Paget's disease the calcium metabolism has been demonstrated to be in positive balance and improvement has been noted following administration of parathormone (Wilder and Howell, and Colt and Lyall). A case reported by Bassler seems to sustain this assumption, his patient showing great symptomatic relief as well as objective evidence of improvement demonstrable in roentgenograms of the bones. Van Hazel and Andrews from extensive metabolic studies also concluded that Paget's disease resulted from hyposecretion of the parathyroid glands and suggested parathormone therapy. Greenwald and Gross advanced the hypothesis that the calcium excretion took place faster than the calcium dissolution in the bone and thus explained the lowering of the abnormally high blood calcium by parathormone in certain cases.

3. Osteomalacia.

Of the skeletal diseases, osteomalacia is most often mistaken for hyperparathyroidism. It is necessary to define the meaning of "osteomalacia" (adult rickets) because the term is often used very loosely. It is a disease in which bone shows widened osteoid seams as a result of failure of calcium deposition in the osteoid "tissue" and is due to deficiency of vitamin D. It is practically non-existent in this country, except in association with fatty diarrhea and the resulting lack of absorption of the fat-soluble vitamin D. The prevalence of the disease in women is possibly explained by the influence of pregnancy and lactation. The bones bend, rather than fracture, and show the radiographic evidence of decalcification but no cysts. The histological appearance of the bone, if decalcified in such a way as to show the osteoid tissue, is pathognomonic. The blood chemistry determinations show a normal or low value for calcium, a corresponding phosphorus figure, and high phosphatase activity. Accordingly, Maxwell finds that tetany is common although usually not severe. The condition responds promptly to antirachitic measures as good food, the administration of cod liver oil, irradiated ergosterol and ultraviolet light. In contradistinction to this Johnson showed experimentally that vitamin D concentrates intensified the disease of hyperparathyroidism. While the hypertrophy of the parathyroid glands in conditions of vitamin D deficiencies is compensatory Erdheim reports, and is certainly a secondary phenomenon, all data indicate that the tumors of the parathyroid glands found in association with osteitis fibrosa cystica are of primary significance etiologically.

4. Focal Osteitis Fibrosa (Solitary Cysts; Local Osteodystrophia Fibrosa; Localized von Recklinghausen's Disease).

Focal osteitis fibrosa, in which one or more bones may be affected with localized changes exactly similar histologically to those of the generalized disease, must be considered in the differential diagnosis. Abel, Thompson and Hawkle state that this localized disease is of more common occurrence and chiefly affects young individuals (ages of five to fifteen years)

whose health is otherwise normal. The proximal metaphyses of the humerus, femur, or tibia are the common sites of its localization (Livingston), and the condition is usually not disabling until spontaneous fractures occur. It has been speculated by Geschickter and Copeland that years elapse before these lesions are observed, and the tendency to spontaneous arrest probably explains why these "healed cysts" are often discovered only incidentally. The slow progress together with the normal radiographic appearance of the remaining skeleton, the normal quantity of calcium, phosphorus and phosphatase of the blood exclude such localized cysts from the category of hyperparathyroidism. D'Auncy and Connell also state,

> We do not agree with Virchow's original viewpoint that these cysts arise in degenerating chondromata. It is noteworthy in this respect that all case reports and pathological discussions of osteitis fibrosa stress the absence of cartilage. The attempts to correlate the localized cystic bone condition in some of their essential details with diffuse fibrocystic disease of bone (von Recklinghausen's disease), other parathyroid disturbances and disorders of calcium-phosphorus metabolism in general hardly seem plausible.

According to Cotton there is definite evidence that giant cell tumors do degenerate to form cysts, and transitional forms are not rarely encountered, but giant cell tumors may be associated with cysts in the same individual and both may occur in hyperparathyroidism. Localized fibrocystic disease not infrequently involves two or more bones in the same individual simultaneously or at different periods without at any time showing a disturbance in calcium metabolism or a generalized demineralization of the skeleton.

5. Polycystic Osteitis Fibrosa.

Geshickter and Copeland consider these lesions as an early stage of the disease described. The small cysts at a later date fuse to form the large solitary cysts.

6. Giant Cell Tumors.

These tumors known under the various titles of giant cell tumor, myeloid sarcoma, or osteoclastoma are somewhat of a histological and clinical enigma. Albright, Aub and Bauer and others have shown that giant cell tumors apparently identical may be completely localized isolated lesions, or be a part of the general osseous involvement of hyperparathyroidism and, if the former, the remaining skeleton and the calcium and phosphorus content of the blood will all be normal. One of us (J. D. B.) has recently produced a giant cell tumor experimentally in a rabbit as a result of trauma which supports the belief of Geshickter and Copeland that these tumors are chronic resorptive lesions resulting from a disturbance of blood supply. Ballin states that

> breaking down of the bony tissue will cause small hemorrhages and granulation tissue with giant cells in response to the irritation in the bone; in all, a picture of so-called brown tumor. This interesting and important factor, namely, that seemingly neoplastic giant cell tissue produced by endocrine gland pathology can be cured by removal of the pathological glands may, in the future, have some influence on the teaching of the origin of some tumors.

Thus, in contradistinction to the concept that giant cell tumors are neoplastic, Ballin and Morse believed that the term "giant cell sarcome" was a misnomer, that it is not a neoplasm, but rather a reaction of osseous tissue to certain factors, such as decalcification in hyperparathyroidism, as verified by the fact that in these cases the tumors often disappear with the recalcification of the skeleton which follows parathyroidectomy.

It is apparent that the occurrence of a giant cell tumor in any individual patient demands differential diagnostic investigation to determine whether it is an isolated entity or only a manifestation incidental to hyperparathyroidism.

7. Osteitis Tuberculosa Multiplex Cystica.

Vastin and Bacon report a multiple cystic state occurring in the diaphysis of

the long bones which simulates osteitis fibrosa cystica roentgenologically. However, this condition occurs in children, the blood chemistry is normal, the tuberculin reaction is usually strongly positive, and finally, a biopsy specimen is diagnostic.

8. Osteogenesis Imperfecta (Fragilitas Ossium).

Although the osteoporosis of the bones in this disease is not unlike that found in the early cases of hyperparathyroidism, the histological osseous picture, the normal levels of calcium and phosphorus of the blood serum, the normal to slightly elevated phosphatase, and principally, the earlier age of onset (infancy) should leave no difficulty in diagnosis. Kaplan, among others, considers this disease to be an endocrine disturbance. It is interesting to note recent reports of the favorable therapeutic response to an ovarian extract in several series of cases of fragilitas ossium.

9. Xanthomatosis Generalisata Ossium.

There exists a generalized xanthomatosis of the bones without symptoms of Schüller-Christian's disease, i.e., without craniohypophyseal localization. The roentgenograms of the osteoporotic lesions of generalized xanthomatosis of the bones may easily be confused with osteitis fibrosa cystica generalisata (Snapper and Parisel). The differentiation can be made by studies of calcium metabolism.

10. Multiple Myeloma.

This condition may very closely resemble hyperparathyroidism. In both, there is often a history of rheumatism, weakness, spontaneous fracture, nephritis or nephrosis often complicated by gastric disturbances, kyphosis, paralysis, with remissions and occurrence in individuals past middle age. Even the *x*-ray findings may closely resemble osteitis fibrosa cystica, as may the presence of hypercalcemia. However, the associated high serum phosphorus and only slightly elevated phosphatase plus the presence of Bence-Jones proteinurie (in 60 to 70 per cent of cases) usually suffice to make the differential diagnosis. In doubtful cases, the biopsy clinches the diagnosis, showing the multiple plasma cell types, the multiple endothelial cell types, and the myelocytic and erythroblastic cell types (Livingston).

11. Metastatic Malignancy.

Skeletal metastases from carcinomas of the prostate, breasts, bronchi, thyroid and from the hypernephromata occasionally cause confusion. However, the differentiation can be made by the presence of hyperphosphatemia as well as hypercalcemia and the fact that the metastases, although often widely disseminated, do not involve the entire skeleton. Although osteolytic as well as osteoplastic, the lyses is obviously that of destruction. It is differentiated also by the other evidences of malignant disease and by the presence of the primary tumor if demonstrable.

Other conditions which have been mistaken for hyperparathyroidism are osteosarcoma (Beck reports three cases in which disarticulations at the hip joint had been performed), chondromata, slipping epiphyses and leontiasis ossea (Cantarow).

With no apparent justification an etiological diagnosis of hyperparathyroidism has been invoked to explain melorheostosis ("candle-drip" hyperostosis of bone), hypophyseal diostosis (Schüller-Christian syndrome), hyperostotic lumbarthria, calcification of the nucleus pulposus, Kümmell's disease, Sicard's disease, Putti's disease, and osteosclerosis fragilis generalisata (osteopoikilosis or "marble bone") (Ballin and Morse, and Moreau). Likewise ankylosing polyarthritis has been attributed to hyperparathyroidism for reasons that follow:

Von Oppel of the University in Leningrad, where men like Bechterew through their work on arthritis had created a background for such studies, brought out the fact that the usual infectious arthritis does not lead to ankylosis, but as soon as hypercalcemia exists simultaneously, calcium deposits around the inflammed arthritic synovia will take place and lead to ankylosis. He, therefore, advised parathyroidectomy (unilateral) in

cases of arthritis with symptoms of parathyroidism, but never recommended it as a cure of arthritis. In other words, if a patient with ankylosing arthritis has hypercalcemia or roentgenographic evidence of decalcification or obvious muscular hypotonia, a parathyroidectomy was done. Oppel first reported favorable results in cases of spondylitis deformans (Strumpell-Marie type) following parathyroidectomy. Later Funsten stated that various types of arthritis besides the polyarthritic ankylosing type showed improvement following parathyroidectomy, but he presented no metabolism studies on these cases showing alterations in the calcium and phosphorus content of the blood.

Bauer has never seen any findings in patients with either form of arthritis that would suggest the existence of hyperparathyroidism as a causal factor. It is true that many of the cases of rheumatoid arthritis show *x*-ray evidence of decalcification, but this is no more than could be accounted for by disuse atrophy. The improvement in cases reported may have been due to anesthesia, rest in bed, or the natural remission of disease. Bauer also states that in the last five years at the Massachusetts General Hospital the two diseases, hyperparathyroidism and ankylosing polyarthritis, have never been observed in the same patient, although seven cases of hyperparathyroidism had been studied. Also in reviewing the literature of reported cases of osteitis fibrosa cystica, we have found no proven cases in which an accompanying arthritis of the ankylosing type was present. However, in many cases the earlier symptoms were often diagnosed arthritis of one sort or another before the true syndrome of hyperparathyroidism was discovered (see Symptoms). The occasional finding of enlargement of the parathyroid glands is probably simply compensatory hyperplasia.

Basophilic adenomas of the pituitary gland (Cushing's disease) may give rise to an osteoporosis as pointed out by Cushing that may resemble von Recklinghausen's disease. Collip shows that there is some evidence that a secondary hyperparathyroidism is present in these cases. In one reported by Cushing the serum calcium was normal and the phosphorus low (2.7 mgms. per cent) and there was a high calcium excretion in the urine, but there was also the obesity, hirsutism, amenorrhea and hypertension characteristic of the basophilic syndrome.

B. The Muscular System in Differential Diagnosis.

In considering the effect of hyperparathyroidism on the muscular system, one is really considering the effect of hypercalcemia per se, i.e., hypotonia. The extreme muscular weakness of these patients may suggest the diagnosis of pseudohypertrophy of the muscles or other muscular dystrophies. Studies of the calcium and phosphorus balance and of the skeleton should determine this point. The cardiac musculature may be involved sufficiently to give symptoms and bizarre electrocardiograms. The smooth musculature of the gastrointestinal tract loses its tone giving symptoms of vague pains, constipation, nausea and vomiting, and indigestion, so that a diagnosis of gastroptosis is often made, or organic diseases of the gastrointestinal tract suggested. Peripheral vasospastic phenomena, such as bluish color of the limbs, has led to a suspicion of Raynaud's disease. Thus it should be remembered that generalized muscular weakness, bizarre gastrointestinal symptoms, and vasospastic phenomena do occur as symptoms of hyperparathyroidism.

C. The Renal System in Differential Diagnosis.

The polydipsia and polyuria resulting from increased calcium and phosphorus excretion in the urine may suggest diabetes mellitus and even insipidus. Renal colic and hematuria or persistent albuminuria often occur, so that a differential diagnosis involves the various nephritides, nephroses and infections of the urinary tract as well as calculi unassociated with parathyroid disease.

PROGNOSIS

Whereas the disease of hyperparathyroidism may produce a fatal issue, either in the form of acute parathyroid poisoning or, more commonly, through renal involvement, it probably smolders for years in the majority of cases, crippling but not killing. Spontaneous remissions are recorded and suggest that hyperparathyroidism may sometimes show relapses and remissions analogous to those seen in acromegaly and exophthalmic goiter.

TREATMENT

1. Medical Treatment.

Albright, Bauer, Claflin and Cockrill report a series of cases treated by ingestion of phosphates with some beneficial results, but advised it only until surgery is available.

Leopold reported apparent recovery of some cases by administration of calcium gluconate (1.5 gm. a day), together with a high calcium diet, rest and massage.

As regards prophylactic therapy for prevention of renal damage in hyperparathyroidism, Albright, Baird, Cope and Blomberg pointed out that fluids should be forced and alkaline urine should be avoided, that the high phosphate diet, prescribed in the foregoing, while indicated for the demineralization process, imperils the kidneys and should be used only when the blood values can be carefully followed and this applies to the high calcium diet as well.

Vitamin D in the form of viosterol in large amounts has been administered to patients but no appreciable effect on the calcium and phosphorus balance was noted by Albright, Aub and Bauer. Johnson states that "whereas vitamin D effectively protects against rickets or osteomalacia, it is evident from the experiments that it intensifies the disease produced by excessive parathyroid hormone." However, Snapper and Boever report that such treatment increased the density of the bones as judged by roentgenograms.

2. *X*-Ray Treatment.

Cutler and Owen believe that radiation therapy is indicated in cases of hyperparathyroidism only under certain circumstances:

a. When there are contraindications to operation;

b. As a postoperative method when an adenoma is only partially removed;

c. When, following removal of one parathyroid adenoma, a persistently high serum calcium suggests the possibility of hyperplasia or adenoma in the glands on the opposite side.

The normal parathyroids are highly resistant to radiation, as has been shown experimentally and verified clinically. After exposure in treatment of the thymus, thyroid, or in cases with carcinomas of the larynx, no tetany develops even after intensive irradiation.

That active hyperplastic cellular tissues are more radio-sensitive is a well established fact attested by the efficiency of this form of therapy in influencing the salivary, ovarian, pituitary or thyroid functions. Merritt and McPeak believe that it would seem likely that adenomatous and hyperplastic parathyroid glands might respond similarly. Unfortunately, this is not the case, because irradiation has effected no cures or even temporary aid. Albright, Aub and Bauer followed the total calcium and phosphorus metabolism over a long period of time in patients who received large doses of *x*-ray over the parathyroid region without detecting any changes in the urinary excretions or blood values and subsequently a parathyroid tumor with no degenerative changes was removed. Similarly, irradiation of the skeleton has little or no benefit upon the tumors, or cysts and merely tends to increase the fibrosis of the marrow and thus enhance the anemia.

3. Surgical Treatment.

Since Mandl's pioneer operation nine years ago, over 100 reported cures of hyperparathyroidism by means of surgical removal of parathyroid adenomas have accumulated. The identification and re-

moval of definite parathyroid tumor is not difficult unless it has an aberrant location. Recently Lahey has suggested a routine for completely exploring the regions in which parathyroid bodies may be found. Brewer has reported the occurrence of parathyroid tissue in the thymus in 4 cases of routine autopsies at the University of Michigan. In Churchill's celebrated case, cure was effected after removal of an adenoma of an aberrant parathyroid gland located in the mediastinum. The mediastinum was searched because the disease, after several explorations of the neck during which parathyroid tissue was removed but no adenomata found, was uninfluenced and progressed, indicating the presence of hyperfunctioning parathyroid tissue in an abnormal location.

Upon the assumption that hyperparathyroidism can exist in the absence of gross hyperplasia or neoplastic changes in the glands, Ballin and Morse have removed normal parathyroids when no tumor was found and have reported evidence of clinical improvement in a group of cases which, as stated previously, do not conform to the classical concept of hyperparathyroidism.

Any actual surgical treatment of the cysts themselves may be necessary after the parathyroidectomy because the fibrous replacement of bone in these cysts may interfere with adequate recalcification and they may persist but tumors (osteoclastomata) disappear. Speed has advocated (a) the cystic area be widely opened, curetted and swabbed out, being left open for invasion by granulation tissue, or (*b*) placing into the cavity transplants of bone from some autogenous source, or, (c) the immediately surrounding cortex be jammed down into the cavity to obliterate it and act as a graft.

POSTOPERATIVE COURSE

Following the removal of a parathyroid tumor, there is often temporarily a marked decrease in the output of urine and in the excretion of calcium and phosphorus. The patients may become practically anuric. Tetany may develop if the blood calcium falls much below 7 or even 8 mgms. per cent. The sudden change from a high level of blood calcium to a low one may cause visual disturbances (temporary hyperacusis) and may affect the mental equilibrium. Such patients may become temporarily apprehensive. The treatment of tetany is beyond the scope of this paper, but one should mention the use of calcium and parathormone as routine postoperative treatment. The time required for the blood calcium and phosphorus to return to normal probably depends mainly on the need of skeletal repair.

The improvement in symptomatology following operation is extraordinary. Whereas the only complaint before operation may have been related to a fracture, the patient not infrequently says after the operation that he has not felt so well in years. There is a rapid gain in weight, constipation disappears, lassitude is replaced by a feeling of energy, bone pain and tenderness disappear within a few days. This rapid disappearance of skeletal symptoms is especially surprising in view of the fact that the radiability is often not appreciably changed for six months. The osteoclastomata gradually disappear, but the cysts may remain indefinitely, as verified by Albright, Aub and Bauer. The renal stones persist and if they give rise to symptoms, should be treated as are calculi unassociated with hyperparathyroidism. Osteoplastic operations may be necessary for correction of deformities resulting from fractured and twisted bones.

CONCLUSION

The diagnosis of hyperparathyroidism must be considered in patients presenting the most varied symptomatology—the patient with widespread skeletal disease with cysts, tumors, and fractures; the patient with an epulis of the jaw or generalized aches and pains varying from pain in the back to flat feet; the individual with renal colic; the patient with unex-

plained polyuria; the uremic; even perhaps the supposedly neurasthenic individual.

Although suspected from other evidence, the final diagnosis is made in the chemical laboratory. Roentgenograms can only add confirmatory evidence. One must think of the diagnosis very often, but be prepared to be disappointed almost, but not quite, equally often.

REFERENCES

Abel, I., Thompson, G. and Hawksle, L. Generalized Osteitis Fibrosa. *Lancet*, 526–529, September 2, 1933.

Albright, F. Hyperparathyroidism. *Med. Clin. of North American*, 18: 1109–1116, Jan., 1935.

Idem. Hyperparathyroidism, its Diagnosis and Exclusion. *New Eng. J. of Med.*, 209: 476, 1933.

Albright, F., Aub, J. and Bauer, W. Hyperparathyroidism. *J. A. M. A.*, 102: 1276–1287, April 21, 1934.

Albright, F., Baird, P. C., Cope, O. and Blomberg, E. Studies on the Physiology of the Parathyroid Glands. *Am. J. Med. Sc.*, 187: 49–52, Jan., 1934.

Albright, F., Bauer, W., Claflin, D. and Cockrill, J. R. Studies in Parathyroid Physiology. *J. Clin. Invest.*, 11: 411–413, 1932.

Ballin, M. Parathyroidism. *Am. Jour. Surg.*, 24: 36–41 (April) 1934; Parathyroidism in reference to orthopedic surgery. *Jour. Bone and Joint Surg.*, 15: 121–134, 1934.

Ballin, M. and Morse, P. F. Parathyroidism. *Am. Jour. Surg.*, 12: 403–416 (June) 1931; Parathyroidism and parathyroidectomy. *Ann. Surg.*, 94: 592–610 (Oct.) 1931.

Bassler, A. An Interesting Result from the Use of Parathyroid Extract in a Case of Osteitis Deformans (Paget's Disease). *J. A. M. A.*, 87: 96–99, July, 1926.

Bauer, W. Hyperparathyroidism. *Jour. of Bone and Joint Surg.*, 15: 135–141, 1933.

Beck, A. Ostitis Fibrosa. *Arch. f. Klin. Chir.*, 152: 123, 1928.

Bodansky, A. Determination of Plasma Phosphatase. *Proc. Soc. Exper. Biol. and Med.*, 28: 760–764, April, 1931.

Bodansky, A., Blair, J. and Jaffe, H. L. Experimental Hyperparathyroidism in Guinea Pigs, Leading to Osteitis Fibrosa. *Jour. Biol. Chem.*, 88: 629–634, 1930.

Brewer, L. A. The Occurrence of Parathyroid Tissue within the Thymus. *Endocrinology*, 18: 397, June, 1934.

Cantarow, A. Parathyroid Tumor. *International Clinics*, 1: 235–241, March, 1934.

Collip, J. B. Diabetogenic, thyrotropic, adreneotropic and parathyrotropic factors of the pituitary. *J. A. M. A.*, 104: 916, 921 (March 16) 1935.

Colt, G. H. and Lyall, A. A case of osteitis deformans treated with parathormona. *Brit. Med. Jour.*, 2: 10–14, 1933.

Cotton, F. J. Discussion on Giant Cell Tumor. *Surg. Gynec. and Obst.*, 60: 329–330, Feb., 1935.

Cushing, H. Further notes on pituitary basophilism. *J. A. M. A.*, 99: 281, 286 (July) 1932.

Cutler, M. and Owen, S. Treatment of Generalized Osteitis Fibrosa Cystica. *Surg. Gynec. and Obst.*, 59: 81–89, July, 1934.

D'Auncy, R. and Connell, J. H. Osteitis Fibrosa Localisata of the Patella. *Jour. of Bone and Joint Surg.*, 32: 689–691, 1934.

Erdheim, J. Ueber Epitherlorperbefunde bei Osteomalacie. Sitzungsb. d. Akad. d. Wiss. Wien. 116–311, 1907.

Funsten, R. W. Certain Arthritic Disturbances Associated with Parathyroidism. *Jour. of Bone and Joint Surgery*, 15: 112–119, 1933.

Geshickter, C. F. and Copeland, M. M. Tumors of Bone. *Am. Jour. of Cancer*, Lancaster Press, 243–332, 1931.

Geshickter, C. F., Copeland, M. M. and Bloodgood, J. C. Osteitis Fibrosa and Giant Cell Tumors. *Arch. of Surg.*, 19: 169–271, 1929.

Greenwald, I. and Gross, J. The effect of the administration of a potent parathyroid extract upon the excretion of N, P, Ca and Magnesium. *Jour. Biol. Chem.*, 66: 216, 1925.

Hannon, R. R., Shorr, E., McClellan, W. S. and DuBois, E. F. A Case of Osteitis Fibrosa Cystica (Osteomalacia), with Evidence of Hyperactivity of the Parathyroid Bodies. *Jour. of Clin. Invest.*, 8: 215, 1930.

Hess, A. F. and Bills, C. E. The Serum Calcium in the Female Cod during the Spawning Season. *Proc. of the Soc. of Exper. Biol. and Med.*, 25: 349, 1927–1928.

Hunter, D. and Aub, J. C. Lead Studies IV. The effect of the parathyroid hormone on the excretion of the lead and of calcium in patients suffering from lead poisoning. *Quart. Jour. Med.*, 20: 123–131, 1927.

Hunter, D. and Turnbull, H. Hyperparathyroidism. *Brit. Jour. Surg.*, 19: 203–284, 1931–1932.

Johnson, J. Experimental chronic hyperparathyroidism. *Am. Jour. Med. Sc.*, 183: 667–684 (June) 1932.

Kaplan, E. B. Multiple Fractures Associated with Blue Sclera. *Jour. of Bone and Joint Surg.*, 32: 629, 1934.

Kay, H. D. Phosphatase in Growth and Disease of Bone. *Phys. Reviews*, 12: 384, 1932.

Leopold, L. Functional Hyperparathyroidism. *J. A. M. A.*, 102: 1536, May, 1934.

Livingston, S. K. Demineralization of the Skeleton. *Am. J. of Surg.*, 27: 464–475, March, 1935.

Maxwell, J. P. Further Studies in Osteomalacia. *Proc. Roy. Soc. Med.*, 33: 19, 1929–1930.

Merritt, E. A. and McPeak, E. M. Roentgen Therapy of Hyperparathyroidism. *Am. J. of Roent.*, 32: 72–81, July, 1934.

Moreau, L. Considerations Radiologiques sur Quelques Affections Osseuses. *Bull et Mem. Soc. de Radiol. Med. de France.*, 19: 471, 1931.

Oppel, W. A. Parathyroid-ectomy for Ankylosing Polyarthritis. *Ann. Surg.*, 90: 978, 1929.

Pemberton, R., John deJ. and Geddie, K. B. Hyperparathyroidism. *Ann. Surg.*, 92: 202–211, 1930.

Perlman, R. Sarcoma Formation in Paget's Disease. *Jour. of Bone and Joint Surg.*, 32: 603–615, 1934.

Richardson, E. P., Aub, J. C. and Bauer, W. Parathyroidectomy in Osteomalacia. *Ann. Surg.*, 90: 730, 1929.

Riddle, O. and Dotti, L. Action of Parathyroid Hormone in Normal and Hypophysectomized Pigeons. *Proc. of the Soc. for Exper. Biol. and Med.*, 32: 507 (December) 1934.

Riddle, O. and Reinhart, W. H. Parathyroid Dysfunction. *Am. Jour. of Physiol.*, 76: 660, 1926.

Rosedale, R. Fibrocystic Disease of Bones. *Am. Jour. of Path.*, 8: 745–754, 1932.

Rowntree, L. G. Hyperparathyroidism, Report of a Case. Proc. Staff Meet. Mayo Clin., 6: 684–688, Nov., 1931.

Schmorl, G. Demonstration. Gesell. f. Natur. Heilkunde, Munschen, Med. Wchnschr., 59: 2891, 1912.

Snapper, I. Parathyroid Tumor and Changes of the Bones. *Arch. Internal Med.*, 16: 506, 1930.

Snapper, I. and Boeve, H. J. Ueber Osteomalacie Deut. *Arch. f. Klin. Med.*, 170: 371, 1931.

Snapper, I. and Parisel, C. Zanthomatosis Generalizata Ossium. *Quart. Jour. of Med.*, 2: 407, July, 1933.

Speed, K. Parathyroidism with Multiple Areas of Cystic Bone Change. *Surg. Clin. of North A.*, 14: 859–868, Aug., 1934.

Taylor, N. B., Weld, C. B. and Sykes, J. F. The Relation of the Parathyroid Glands to the Action of Irradiated Ergosterol. *Proc. Roy. Soc. B.*, 116: 10–26, 1934.

VanHazel, W. and Andrews, E. Hyperparathyroidism. *Surg., Gynec., and Obst.*, 45: 54, July, 1927.

Vastine, J. H. and Bacon, E. P. Osteitis Tuberculosa Multiplex Cystica. *Radiology*, 24: 22–30, Jan., 1935.

Wilder, R. M. and Howell, L. P. Etiology and diagnosis in hyperparthyroidism. *J. A. M. A.*, 106: 427 (Feb. 8) 1936.

Addison's disease (melasma suprarenale) was named after Dr. Thomas Addison (1793–1860) of Guy's Hospital, London, England.

Banti's disease, characterized by cirrhosis of the liver, hypertrophy of the spleen, and ascites, was named after Guido Banti (1853–1925) of Florence, Italy.

Basedow's disease (exophthalmic goiter) was named after the German physician, Karl A. von Basedow (1799–1854), who described it in 1840.

The name of Bell's disease or palsy (facial paralysis) is derived from that of Sir Charles Bell (1774–1842), a Scotch physiologist who lived in London.

HYPERPARATHYROIDISM

COLLECTED CASES

J. E. JACOBS, M.D. AND J. DEWEY BISGARD, M.D.

OMAHA, NEBRASKA

IN two previous articles* the history, etiology, pathology, diagnosis and treatment of hyperparathyroidism were discussed. Brief summaries of cases collected from the literature follow.

To 1931 there had accumulated in the literature 101 proved cases of hyperparathyroidism. Ninety-seven of these were collected and abstracts reported in the British Journal of Surgery by Hunter and Turnbull.† Seven cases not included in their report are abstracted here. In this collection there were included only those cases in which there was found either a tumor of a parathyroid body at operation or necropsy or an unquestionable negative balance in calcium metabolism. Thus to date there has been reported at least 145 proved cases of hyperparathyroidism.

CASE REPORTS

CASE I (Schnabel, T. G., *Med. Clin. N. Amer.*, 14: 977–988 [Jan.] 1931). A white male with marked deformity from bowing of the extremities, stated that five years previously he received a fracture of the mandible while boxing. Biopsy of the jaw was reported pathologic giant cell tumor. Besides increasing deformity of the spine and legs, there has been generalized body pain, urinary frequency, nausea and vomiting. The urine had contained two plus albumin, the serum calcium was 15.8 mgms. per cent, and phosphorus 2.7 mgms. per cent. There was generalized osteoporosis and a right renal calculus. A hyperplastic parathyroid gland measuring 2 cm. × 2 cm. was removed on the left side. There was persistence and recurrence of symptoms and at reoperation another parathyroid adenoma, 3.5 × 2 × 1.5 cm. was found on the same side at a deeper level. The patient made a complete recovery.

CASE II (Ballin, M. and Morse, P. F. *Am. Jour. Surg.*, 12: 403–416 [June] 1931). A female, 39 years old, in 1914 developed a swelling in the left forearm (a bone cyst). In 1915 she could not walk because of pain in the spine and legs. She died in 1925 and autopsy showed that all the bones were thickened, cystic and cut easily; also a left parathyroid tumor was found.

CASE III (Ballin, M. and Morse, P. F. *Ann. Surg.*, 94: 592–609 [Oct.] 1931). A male, 65 years old, with onset of symptoms in 1927, complained of backache, pain in the legs, progressive curvature of the spine and height diminished two inches. A diagnosis of metastatic malignancy was made. *X*-ray examination showed generalized osteoporosis with compression of the lower lumbar vertebra. He was entirely bedfast. Serum calcium was 14.8 mgms. per cent. A right thyroparathyroidectomy was done, removing two cystic adenomatous parathyroid glands followed with complete recovery.

CASE IV (Ballin, M. and Morse, P. F. *Ann. Surg.*, 94:592–609 [Oct.] 1931). A male, 55 years old, in 1930 noticed onset of backache and progressive disability. Radiographs showed general demineralization of the skeleton with compression fracture of the lower thoracic segments. The serum calcium was 18.6 mgms. per cent. Three hyperplastic parathyroid bodies were removed, and patient made a good recovery.

CASE V (Ballin, M. and Morse, P. F. *Ann. Surg.*, 94:592–609 [Oct.] 1931). A female of 57 years, in 1927 developed backache with kyphosis and shortening of the spine. *X*-ray examination showed general demineralization of the skeleton, the serum calcium was 14.6 mgms. per cent. Two adenomatous parathyroid bodies were removed, and the patient recovered entirely.

CASE VI (Dresser, R. and Hampton, A. O. *Am. Jour. Roentgenol. and Radium Therap.*, 25:739–748, [June] 1931). A female, 44 years

* JACOBS, J. E. and BISGARD, J. D. *Am. Jour. Surg.*

† HUBBARD, R. S. and WENTWORTH, J. A. A case of metastatic calcification associated with chronic nephritis and hyperplasia of the parathyroids. *Proc. Soc. Exper. Biol. and Med.*, 18: 1307, 1921.

old, complained of rheumatism with pain and swelling of the right forearm, frequency of urination, and the loss of fifty pounds. Fourteen years ago, she noticed a swelling in the region of the right lobe of the thyroid gland and was told that she had thinning of the bones. The blood calcium was 13.7 mgms. per cent, and the phosphorus 2.6 mgms. per cent. At operation a tumor the size of a small tangerine (6.5 × 5 × 3 cm.) was removed. Ten days later the blood calcium was 10.1 mgms. per cent and the phosphorus 3.35 mgms. per cent. The patient recovered.

Case VII (Noble, T. P. *Jour. Bone and Joint Surg.*, 14: 181–185 [Jan.] 1932). A Chinaman of 40 years had pain and swelling of the right arm. Roentgenograms showed a pathological fracture of the right humerus with numerous, irregular expansions of the cortex with cystic formation. He later developed a spontaneous fracture of the left femur, then right femur, and swellings of the tibia and fourth rib. The serum calcium was 14.2 mgms. per cent. At operation no tumor was found, but autopsy revealed an adenoma of the parathyroid gland measuring 2 × 1.8 × 0.5 cm.

Case VIII (Gordon-Taylor, G., Wiles, P. and Baker, S. L. *Brit. Jour. Surg.*, 19. 106–618 [April] 1932). A male, 20 years, complained of pain in the left buttock and thigh followed by a spontaneous fracture of the left femur and an area of localized fibrocystic disease. The serum calcium was 16.9 mgms. per cent, and the phosphorus 4.2 mgms. per cent. The lesion was considered a malignant neoplasm and a disarticulation at the hip was done. He was well for about one year, then fractured the right femur and developed a collapse of the eleventh thoracic vertebra. An adenoma of the parathyroid gland was removed and the patient recovered.

Case IX (Wilder, R. M., Camp, J. D., Robertson, H. E. and Adams, M. *Proc. Staff Meet. Mayo Clinic*, 7: 597–606 [Oct. 19] 1932). A female of 48 years, who became nervous at 41, developed stiff knees at 45 years and pain in both knees at 46. She then developed sciatica which confined her continuously to bed. The calcium, phosphorus and blood serum were normal. Two years later, she developed a pathological fracture of the left femur. The phosphatase value of the blood was high. A tumor of the parathyroid gland measuring 2 × 1.5 × 1 cm. was removed from its location between the trachea and esophagus. The patient died later of gastrointestinal symptoms with nutritional edema.

Case X (Babcock, W. W. *Surg. Clin. N. Amer.*, 12: 1387–1392 [Dec.] 1932). A single female, aged 25 years, first developed weakness in the left knee and one year later, swelling about this knee and a dull aching pain. The lesion was diagnosed as a giant cell tumor. There then developed a fusiform swelling of the first phalanx of the right index finger which radiologically had a "soap bubble" appearance and the skull showed a "nigger wool" appearance. There were bilateral renal calculi. The blood serum calcium was 12.5 mgms. per cent. A biopsy from the finger proved to be a giant cell tumor. A cystic tumor of the parathyroid gland measuring 1 cm. in diameter was removed. The patient died a few days later.

Case XI (Landon, J. F. *Jour. Pediat.*, 1: 544 [Nov.] 1932). A white boy, 2½ years old, developed fever, cough and irritability at six months of age, and was treated for swelling of the left jaw at 11 months. He then complained of pain on being handled and limped on attempting to walk. The blood serum calcium varied from 10.5 to 19.4 mgms. per cent and the phosphorus from 2.5 to 5.5 mgms. per cent. *X*-ray films showed cystic areas in all long bones. A small mass was palpated near the left lobe of the thyroid gland. A small adenoma of the parathyroid gland was removed from the left posterolateral aspect of the left lobe of the thyroid gland and the patient made a rapid improvement with cure. No tetany developed.

Case XII (Rosedale, R. S. *Am. Jour. Path.*, 8: 745–754 [Nov.] 1932). A white female, 50 years old, complained of aching muscles, general fatigability, and pain about both knee joints and in the lower part of the right tibia. She was unable to walk on account of weakness. There was also a history of renal calculi and there was a firm mass in the region of the right lobe of the thyroid gland. The blood calcium was 13.82 mgms. per cent, and the phosphorus 2.3 mgms. per cent. There were cystic areas and decalcification in the ribs, clavicles, scapulas, humerii, tibias and mandible. A tumor of one of the parathyroid glands weighing 7 grams and measuring 2 × 2.5 cm. was removed and the patient recovered.

Case XIII (Cohen, H. and Kelly, R. E. *Brit. Jour. Sur.*, 20: 472–478 [Jan.] 1933). A female, 48 years old, had pain and swellings

in the jaw, head and legs. She had had a nephrotomy and four stones had been removed. The blood calcium was 13 to 17.4 mgms. per cent and the phosphorus 2.4 to 2.6 mgms. per cent. A parathyroid adenoma measuring 2.5 × 1.5 × 1 cm. was removed and the patient recovered.

Case xiv (Elmslie, R. C. *Brit. Jour. Surg.*, 20: 379–507 [Jan.] 1933). A female aged 45 years had pain in the left arm on exertion for ten years. Nine years ago she had a fracture of the right humerus and four years ago a spontaneous fracture of the same humerus. *X*-rays showed cysts and decalcification at the fracture site and the area was curetted several times. The blood calcium was 14 mgms. per cent and the phosphorus 1 mgm. per cent. A parathyroid tumor on the left side was removed and the patient recovered.

Case xv (Elmslie, R. C. *Brit. Jour. Surg.*, 20: 379–507 [Jan.] 1933). A female, age 26 years, complained of aching in the bones and stated that an epulis had been removed from the jaw and that there had been a fracture of the femur and bilateral renal calculi. Roentgenogram showed cysts and general skeletal decalcification. A parathyroid tumor was removed and the patient was cured.

Case xvi (Elmslie, R. C. *Brit. Jour. Surg.*, 20: 379–507 [Jan.] 1933). A female, age 23 years, had pain in the left leg and a spontaneous fracture of the left femur followed later by a fracture of the right femur. She then developed swelling of the hands. The blood calcium was 12.7 mgms. per cent and the phosphorus 2.7 mgms. per cent. A parathyroid tumor was removed and the patient recovered.

Case xvii (Rankin, F. W. and Priestly, J. T. *Amer. Jour. Surg.*, 20: 298–314 [May] 1933). A female, 34 years old, complained of difficulty in swallowing, pain in the right hip and generalized body pains. Roentgenograms suggested multiple metastases to the lumbar spine. Later a diagnosis was made of prolonged nutritional deficiency with osteoporosis. With the aid of proper diet, the patient improved and was able to be about for two years, then returned with pain in the right knee and a palpable node at the lower pole of the right lobe of the thyroid gland. The blood calcium was 12.2 mgms. per cent and phosphorus 1.2 mgms. per cent. The basal metabolic rate was plus 21 per cent. A partial thyroidectomy with removal of a parathyroid tumor measuring 6 mm. in diameter was done. The pathological diagnosis was malignant adenoma of a parathyroid gland.

Case xviii (Keynes, G. and Taylor, H. *Brit. Jour. Surg.*, 21: 20–28 [July] 1933). A male, 18 years old, complained of acute abdominal pain with persistent vomiting and urinary frequency. He had passed several small calculi. A year previously he had had pain in the right knee and swelling in the right jaw. The latter was explored and the tissue diagnosed as osteoclastoma. Six years previously he had had aching pain in all bones, headaches, spontaneous fractures of the right humerus and generalized muscular atony. Roentgenograms show a generalized osteitis fibrosa cystica. The blood calcium was 14.7 mgms. per cent and the phosphorus 1.47 mgms. per cent. A cystic parathyroid tumor measuring 3.5 × 2.5 cm. was removed and the patient recovered.

Case xix (Dyke, S. C., Walker, R. M. and Freman, E. *Lancet*, 2: 530–532 [Sept. 2] 1933). A female, aged 14 years, with "rheumatism" and pain in the legs showed in the roentgenograms generalized rarefaction of the skeleton. The blood calcium was 16.7 mgms. per cent. She developed pathological fractures of both femurs and a diagnosis of osteomalacia was made. A tumor of a parathyroid gland, 3 × 2 × 1 cm. on the left side was removed and the patient became well.

Case xx (Dyke, S. C., Walker, R. M. and Freman, E. *Lancet*, 2: 530–532 [Sept. 2] 1933). A female, aged 14 years, complained of a progressive painless deformity of the spine and lower extremities, of four years duration. She developed pathological fractures of the left clavicle and of both bones of the forearm. The blood calcium was 17.7 mgms. per cent and the phosphorus 2.8 mgms. per cent. Roentgenograms showed generalized osteoporosis. A tumor weighing 5 grams was removed and the patient recovered.

Case xxi (Schlesinger, H. and Gold, E. *Klin. Wchnschr.*, 12: 784–787 [May 20] 1933). A female, aged 42 years, developed during postpartum from a miscarriage, severe pains in the legs with anorexia, and was unable to lift the legs. Her stature became gradually shorter. There was an enlargement of the left lobe of the thyroid gland, general decalcification and cyst formation of all bones. The blood serum calcium was 15.8 mgms. per cent and the phosphorus 2 mgms. per cent. A parathyroid adenoma in the left upper pole

of the thyroid gland, measuring 5 × 4 × 4 cm., was removed. The patient recovered.

Case XXII (Abel, A. L., Thomson, G. and Hawksley, L. M. *Lancet*, 2: 525–529 [Sept. 2] 1933). A female, aged 58 years, who had had two children, developed weakness of all her extremities, especially the right arm, and pain in the bones of the left leg. She was always tired and faint. There was a swelling over the anterior aspect of the left tibia. Roentgenograms showed cystic areas in the left tibia and rarefaction of the entire skeleton. A biopsy was typically ostetitis fibrosa. The blood serum calcium was 14 mgms. per cent and the phosphorus 1.8 mgms. per cent. A parathyroid tumor was removed from the lower pole of the thyroid gland and one year later the patient was well.

Case XXIII (Mimpriss, T. W. and Butler, R. W. *Brit. Jour. Surg.*, 21, 500–506 [Jan.] 1934). A male, aged 17 years, first developed pain in both knees and noticed tenderness on pressure in all long bones. He also had an epulis of the jaw. The serum calcium was 18.8 mgms. per cent and the phosphorus 2.9 mgms. per cent. There was an albuminuria. Roentgenograms showed a cystic area in both tibias and in both femurs. A retrosternal parathyroid tumor 12 mm. in diameter was removed from the thymus gland in which it was imbedded. The patient became well.

Case XXIV (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A female, aged 46 years, complained of weakness, constipation, and pain and tenderness in all bones. Roentgenograms showed decalcification and cysts in all long bones. The blood serum calcium was 13.7 mgms. per cent, and the phosphorus 1.9 mgms. per cent. A tumor was palpated in the neck, and this parathyroid adenoma measuring 6.5 × 5 × 3.5 cm. was removed. The patient recovered.

Case XXV (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A female, 60 years old, complained of weakness, constipation, weight loss, backache and polyuria. Roentgenograms showed decalcification of the entire skeleton. The blood serum calcium was 12.3 mgms. per cent and the phosphorus 3.6 mgms. per cent. A right parathyroid adenoma measuring 1 × 7 × .5 cm. was removed and the patient recovered.

Case XXVI (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A female, aged 13 years, complained of weakness, loss of weight, bone pain and polyuria. There was marked decalcification with cyst formation in all bones, and nephrolithiasis. The blood calcium was 12.1 mgms. per cent and the phosphorus 4.7 mgms. per cent. There was albuminuria. A right parathyroid adenoma measuring 2.2 × 1.7 × 1 cm. was removed and the patient recovered.

Case XXVII (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934.) A female, 41 years old, complained of weakness, constipation, bone pain, kyphosis, polyuria. She showed decalcification and cysts in all bones and a pathological fracture of the femur, as well as bilateral renal stones. The blood calcium was 14.2 mgms. per cent and the phosphorus 2.3 mgms. per cent. There was also albuminuria. A parathyroid adenoma 3.5 × 2 × 1 cm. was removed, followed by the patient's recovery.

Case XXVIII (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A female, aged 55 years, had complained of polyuria and renal colic. *X*-ray examination showed nephrolithiasis and mild decalcification of all bones. There was an albuminuria. A parathyroid adenoma on the right side measuring 1 × 1 × 1.5 cm. was removed, and the patient recovered.

Case XXIX (Albright, F., Aub, J. C. and Bauder, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A male, aged 35 years, complained of weakness, constipation, loss of weight, bone pain, polyuria and fractures with deformity. *X*-ray studies showed decalcification and cyst formation, pathological fractures and bilateral nephrolithiasis. The blood calcium was 16.5 mgms. per cent, and the phosphorus 1.4 mgms. per cent. There was an albuminuria. An adenoma of the parathyroid gland, measuring 3 × 3 × 3 cm. was removed from the mediastinum. The patient died one month later following removal of renal calculus.

Case XXX (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A female, 36 years old, complained of weakness, ease of fatigue, constipation, weight loss, pain in the bones, epulis and polyuria. Roentgenograms showed decalcification and cyst formation in all bones, the blood calcium was 13.7 mgms. per cent and the phosphorus 2.4 mgms. per cent. An adenoma of a parathyroid gland measuring 3.5 × 2 ×

1.5 cm. was removed from the mediastinum and the patient recovered.

Case xxxi (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A female, aged 44 years, complained of localized bone pain, and had a pronounced limp. Roentgenograms showed mild decalcification of the skeleton and a right renal calculus. The blood calcium was 14 mgms. per cent and phosphorus 1.5 mgms. per cent. A right parathyroid adenoma measuring 2 × 0.8 × 16 cm. was removed and the patient recovered.

Case xxxii (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April] 1934). A male, 33 years old, complained of weakness, fatigue, bone pain, polyuria and decalcification of the skeleton and bilateral nephrolithiasis. The blood serum calcium was 16.8 mgms. per cent and phosphorus 2.9 mgms. per cent. There was also an albuminuria. A left adenoma of a parathyroid gland measuring 4 × 2.2 × 2 cm. was removed. The patient recovered.

Case xxxiii (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1278 [April 21] 1934). A female, 54 years of age, complained of weakness, loss of weight, a compression fracture of a vertebra with a cord bladder. There was mild decalcification of all bones and a bladder stone. The blood serum calcium was 14 mgms. per cent and phosphorus 2.3 mgms. per cent. A left parathyroid adenoma measuring 3 × 1.7 × .8 cm. was removed with partial recovery of symptoms.

Case xxxiv (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A female, aged 53 years, complained of weakness, fatigue, constipation, weight loss, bone pain and epulis of the jaw and polyuria. There was general skeletal decalcification with cystic areas and pathological fractures. The blood serum calcium was 13.9 mgms. per cent and the phosphorus 1.6 mgms. per cent. There was also albuminuria. A left parathyroid adenoma measuring 3 × 2 × 1 cm. was removed. The patient was much improved.

Case xxxv (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A female, age 51 years, complained of lassitude, bone pain and renal colic. There was no decalcification of the bones but there were bilateral renal calculi. The blood serum calcium was 11.5 mgms. per cent, and the phosphorus 3.5 mgms. per cent. A left parathyroid adenoma measuring 1.5 × 1.5 × 1.5 cm. was removed. The patient recovered.

Case xxxvi (Albright, F., Aub, J. C. and Bauer, W. J. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A male, aged 22 years, complained of weight loss and hematuria. There was no skeletal decalcification but there was a right renal calculus. The blood serum calcium was 15.8 mgms. per cent, and the phosphorus 2.8 mgms. per cent. A left parathyroid adenoma measuring 1.7 × 1.7 × 1.7 cm. was removed. The patient rapidly regained weight and strength.

Case xxxvii (Albright, F., Aub, J. C. and Bauer, W. J. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A male, aged 52 years, was thin, chronically fatigued and complained of renal colic. There was moderate decalcification of the skeleton and calcification in both kidneys. The blood serum calcium was 14.5 mgms. per cent and the phosphorus 1.9 mgms. per cent. A right parathyroid adenoma measuring 2.2 × 1.2 × 1 cm. was removed. The patient made a partial recovery.

Case xxxviii (Albright, F., Aub, J. C. and Bauer, W. J. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A female, aged 62 years, complained of lassitude and polyuria. There was no decalcification but there were bilateral renal calculi. The blood serum calcium was 15 mgms. per cent and phosphorus 2.2 mgms. per cent. Three hyperplastic parathyroid glands were removed.

Case xxxix (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A male, age 26 years, complained of lassitude and polyuria. There was no skeletal decalcification but there were ureteral calculi. The blood serum calcium was 16.7 mgms. per cent and the phosphorus 1.7 mgms. per cent. Two hyperplastic parathyroid glandules were removed.

Case xl (Albright, F., Aub, J. C. and Bauer, W. *J. A. M. A.*, 102: 1276–1287 [April 21] 1934). A female, aged 55 years, complained of renal colic. There was no skeletal decalcification but there was an ureteral calculus. The blood serum calcium was 12.4 mgms. per cent and the phosphorus 2.1 mgms. per cent. Three hyperplastic parathyroid glandules were removed.

Case xli (Bevere, L. and Sorrentino, B. *Riforma Med.*, 48: 313 [Feb. 27] 1932). A female, aged 35 years, during her tenth

pregnancy developed pains in both legs and three years later, spontaneous fractures of the left humerus, right femur, right clavicle and the right humerus. There was decalcification of the bones with cyst formation and a blood calcium of 14.65 mgms. per cent, and phosphorus 2.95 mgms. per cent.

Case xlii (*Jour. de méd. de Par.*, 53: 178–182 [March 3] 1932). A woman, aged 41 years, who had had pain for four years in both legs and the lumbar region, fell and developed a fracture of the right femur which failed to unite. Sometime later there was a spontaneous fracture of the left femur and right humerus. She also had marked muscular atony, constipation, anorexia and vomiting. Her stature had shortened with the development of a kyphosis. All of her teeth had fallen out. The blood Wassermann was positive. Roentgenograms showed generalized fibrocystic disease. The blood serum calcium was 14.5 mgms. per cent and the phosphorus was 4.3 mgms. per cent. Operation was refused and at postmortem examination a tumor was found under the right lobe of the thyroid gland measuring 35 × 30 × 20 mms., which histologically was parathyroid tissue.

Case xliii (*Acta chir. Scandin.*, 74: 485–490, 1934). A man, 74 years old, complained that for two years he had had rheumatic pain, fatigue and an ununited fracture of the neck of the right femur. He developed a spontaneous fracture of the left femur, marked constipation and urinary frequency. Roentgenograms were characteristic of a fibrocystic disease. The blood serum calcium was 13 mgms. per cent. A parathyroid tumor of the left side measuring 15 × 25 × 35 mm. was removed. The patient died three days later but there was no autopsy.

Case xliv (Elson, K. A., Wood, F. C. and Ravdin, I. S. *Am. Jour. Med. Sc.*, 191: 49–55 [Jan.] 1936). A woman, aged 35 years, for five years had complained of lassitude and fatigability, polyuria and polydipsia and constipation, and for two years joint and bone pain. Roentgenograms were characteristic of generalized fibrocystic disease and there was parenchymal calcification of both kidneys. The blood serum calcium was 15 mgms. per cent and the phosphorus 2.7 mgms. per cent. Two parathyroid adenomas were removed and the patient made a complete recovery.

Case xlv (Barker, L. F. *Jour. Bone and Joint Surg.*, 16: 435–440 [April] 1934). A female, aged 65 years, stated that ten years previously she had developed a pathological fracture of the right humerus and a year later a refracture of the same arm. Diagnosed as osteitis fibrosa cystica, the cyst was curetted. Roentgenograms showed extensive rarefaction and cyst formation in all bones and also nephrolithiasis of the left kidney. The blood calcium was 12.9 mgms. per cent and the phosphorus 2.7 mgms. per cent. A right parathyroid adenoma measuring 2.5 × 1.5 cm. was removed and the patient became fairly well.

Case xlvi (Cahill, J. A., Jr. *M. Ann. District of Columbia*, 2: 275–278 [Nov.] 1934). A female, aged 22 years, complained of pain and swelling of the right forearm, nausea and vomiting, and diarrhea for two years. She developed fractures of the left humerus and the left patella. There was a small tumor palpable in the right side of the neck. Roentgenograms were characteristic of generalized fibrocystic disease with pathological fractures. The blood serum calcium was 17.2 mgms. per cent and the phosphorus 1.5 mgms. per cent. A pea-sized tumor was removed from the inferior border of the right lobe of the thyroid gland and the patient recovered. The blood calcium and phosphorus became normal.

Case xlvii (Lahey, F. H. and Haggart, G. E. *Surg., Gynec., and Obst.*, 60: 1033–1051 [June] 1935). A female, 62 years old, with a deformity of the spine complained of diffuse pain throughout the body. Roentgenograms were characteristic of fibrocystic disease. The blood serum calcium was 17.2 mgms. per cent and the phosphorus 2.5 mgm. per cent. A left parathyroid adenoma measuring 1.5 × 3 × 4 inches was removed with relief of symptoms.

Case xlviii (Lahey, F. H. and Haggart, G. E. *Surg., Gynec., and Obst.*, 60: 1033, 1051 [June] 1935). A female, aged 53 years, complained of pain in the knees and in the back of the neck. There was diffuse osteoporosis of the skeleton. The blood serum calcium was 14.9 mgms. per cent and the phosphorus 2.2 mgms. per cent. An olive-sized parathyroid adenoma was removed and the patient improved.

Case xlix (Lahey, F. H. and Haggart, G. E. *Surg., Gynec., and Obst.*, 60: 1033–1051 [June] 1935). A female, 74 years old, complained of severe pain in the legs and gave a history of spontaneous fractures. She had a goiter and roentgenograms were characteristic of general fibrocystic disease. The blood serum calcium was 11.3 mgms. per cent and the phosphorus 1.9 mgms. per cent. A parathyroid adenoma

the size of a lime and another the size of a cherry were removed and the patient recovered.

Case l (Lahey, F. H. and Haggart, G. E. *Surg., Gynec. and Obst.*, 60: 1033–1051 [June] 1935). A female, 44 years of age, complained of diffuse low back pain and pain in both knees for two years and had developed a scoliosis and kyphosis. Roentgenograms were characteristic of generalized fibrocystic disease. The blood serum calcium was 15.1 mgms. per cent and the phosphorus 2.4 mgms. per cent. A right parathyroid adenoma the size of a walnut was removed.

Case li (Lahey, F. H. and Haggard, G. E. *Surg., Gynec. and Obst.*, 60: 1033–1051 [June] 1935). A female, age 52 years, complained of low back pain and generalized pain throughout the body. There was general skeletal decalcification with a compression fracture of the first lumbar vertebra. The blood serum calcium was 14.1 mgms. per cent and the phosphorus 2 mgms. per cent. A right parathyroid adenoma the size of a lima bean was removed and the patient recovered.

SUMMARY

All the reported cases present many or all of the clinical features characteristic of hyperparathyroidism, and in all of those in which surgical or postmortem exploration of the neck was performed definite tumors of one or more parathyroid glands were found. The skeletal changes consisted of osteoporosis, fibrocystic changes, osteoclastomas and spontaneous fractures. All the patients complained of bone pains and weakness, many of constipation, and several of symptoms associated with renal calculi. Diminution in stature and skeletal deformities were noted frequently. In those patients with the studies of blood and the urinary chemistries there were definitely demonstrable, hypercalcinemia, hypophosphonemia, hypercalcinuria and hyperphosphouria.

SURGICAL TREATMENT OF PENDULOUS HYPERTROPHIC BREAST

PALACIO POSSE, M.D.

BUENOS AIRES, ARGENTINA

A NEW method for the treatment of mammary ptosis is described which the author used in 126 recent surgical cases. After three years spent in Europe studying plastic and reconstructive surgery and six years of continuous practice in Argentina with over 600 patients treated, I concluded that the method presented offers a number of advantages, which make it possible to attain the best results without the danger of necrosis.

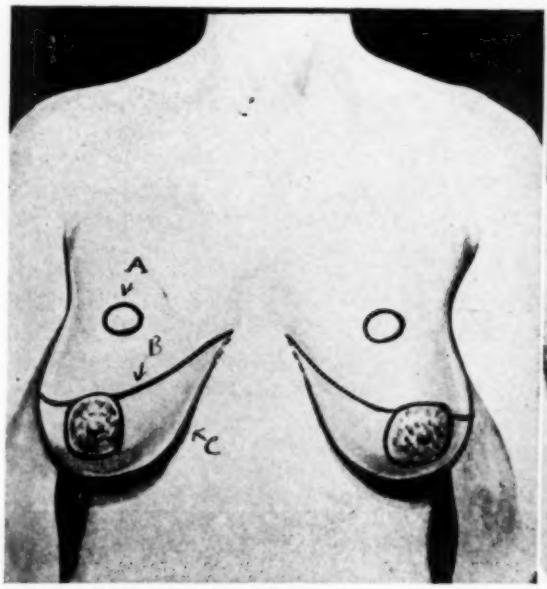


FIG. 1. The operating lines traced on the breasts of the patient; A, the buttonhole where the areola and nipple will be placed in their new position; B, represents the anterior flap and C, the submammary fold. These two lines intersect at their ends.

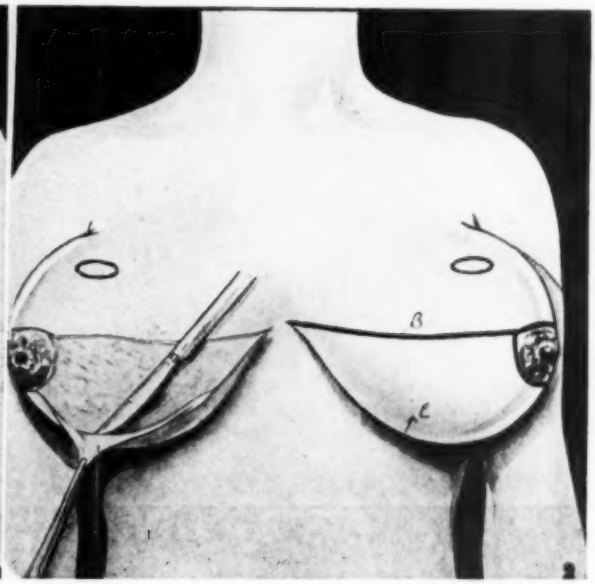


FIG. 2. The patient is lying down horizontally, in which position the gland points outward and upward. The drawing shows clearly the section between lines, B and C, where the epidermis is removed.

PRELIMINARY TREATMENT

Following the usual analysis previous to all operations a light diet for two days is recommended before the operation, giving preference to carbohydrates. The night before the operation, she should retire early and take 1 or 2 grains of veronal or a similar sedative. Shortly before operation another grain of veronal is given and followed by a strong dose of escopolamina-eucodal-efetonina, while tracing the outline of the operation with a dermographic pencil. The skin is disinfected with a solution of two parts alcohol and one part tincture of iodine, or with a solution of picric and alcohol. Special care should be exercised to prevent the traces of the outline of the operation from being wiped out.

OPERATIVE TECHNIQUE

The operation is generally done in one stage under local anesthesia, using less than 200 c.c. of one half per cent solution of novocaine. By this method I have operated upon both breasts at the same time, without ever having to resort to general anesthesia.

By subdividing the operation into five separate stages I shall attempt to describe the method in all cases of pendulous hypertrophic breasts.

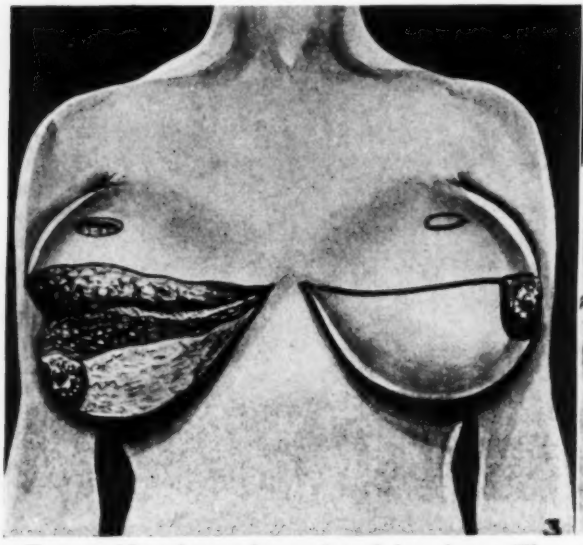


Fig. 3. Also shows the patient lying down. The buttonhole for the areola has been opened and the gland has been cut into two segments by an incision which reaches down to the pectoral fascia.

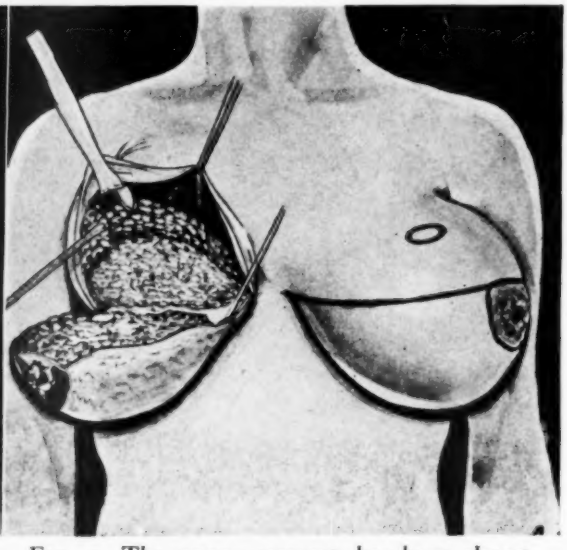


Fig. 4. The upper segment has been almost entirely emptied of the gland, the lower segment to occupy its position.

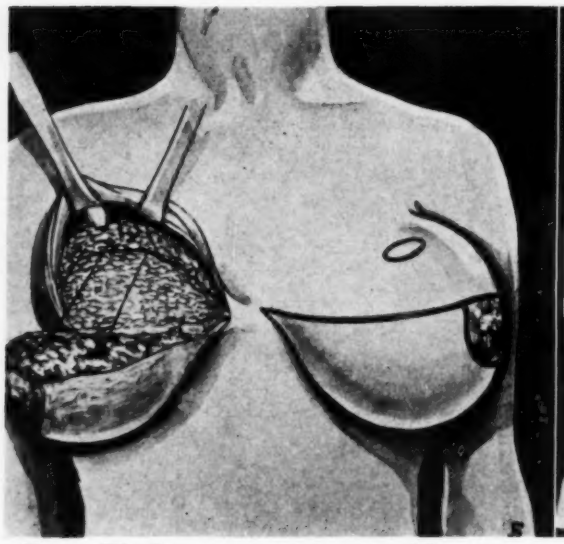


Fig. 5. The lower segment is sutured by two or three silk threads to the pectoral fascia.

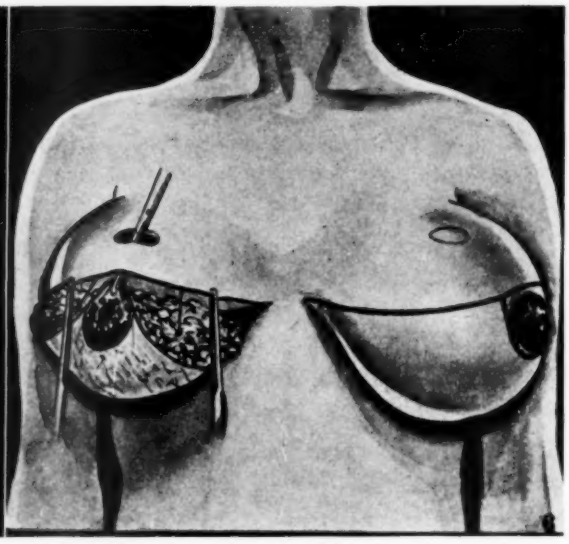


Fig. 6. By means of a pair of forceps, the areola is made to pass through the buttonhole into its new position.

1. Anesthesia is applied superficially to the skin between the lines B and C, Figure 1, and subsequently the epidermis of that section is removed as seen in Figure 2. The object of this procedure is to insure the greatest possible safety to the areola and the nipple, maintaining not only the main blood supply from the interior vessels but also, and to my mind the most important point, the skin blood supply, as represented by the full conservation of the dermis with its nerves and blood vessels.

2. Deep anesthesia of the gland is applied on the whole extension of line B,

after which the gland is divided into two segments by an incision which reaches down to the pectoral fascia. At the same time a buttonhole is shaped as described in Figure 3.

3. The major part of the upper segment of the gland is then emptied as shown in Figure 4 and by means of two or three stitches with thick silk thread the lower segment is fixed to the pectoral fascia and to the remaining portion of the gland of the upper segment (Fig. 5).

4. The nipple is henceforth pulled through the buttonhole into its new position, where it is carefully sutured (Fig. 6).

5. In the last stage the edge of the upper segment following line B has to be thinned out, in order to join the lower segment on line C, where both parts are sutured

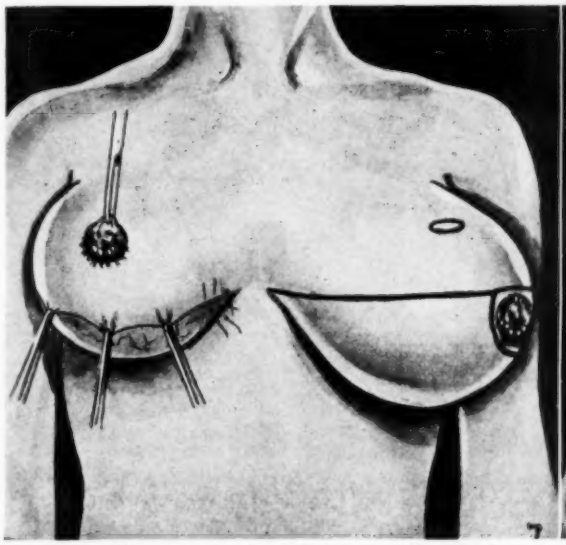


Fig. 7. The entire rim of the areola has been sutured and the anterior flap is being pulled with Chaput pincers and sutured to the submammary fold.

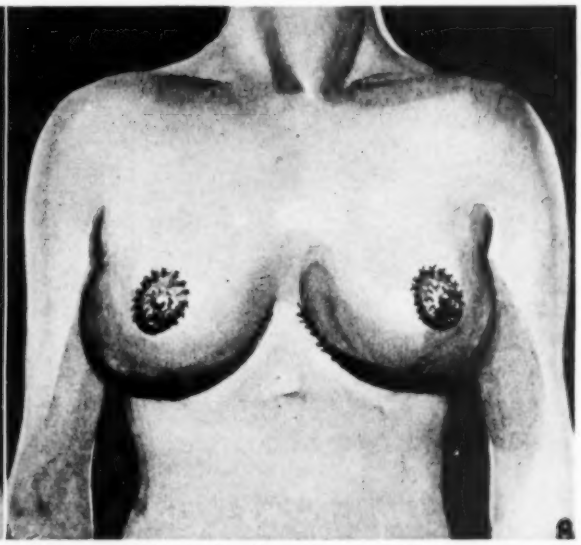


Fig. 8. Areola and submammary fold are sutured; the patient is in the upright position.

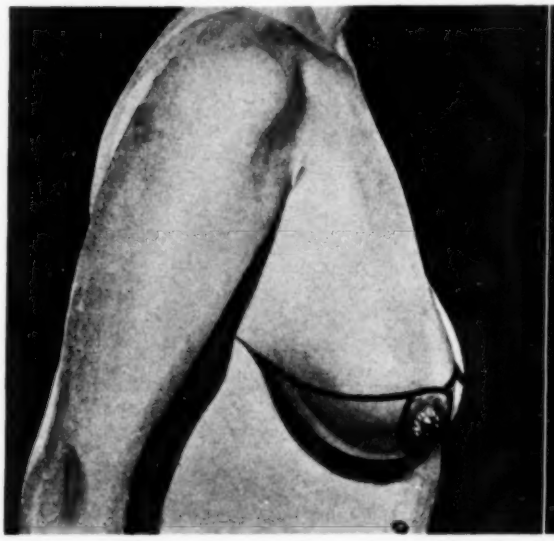


Fig. 9. The patient is shown in profile, the operating outline is traced on the breast and the nipple points downward.

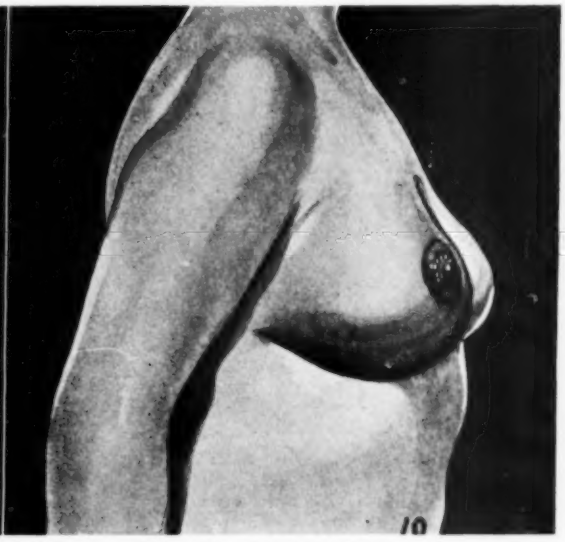
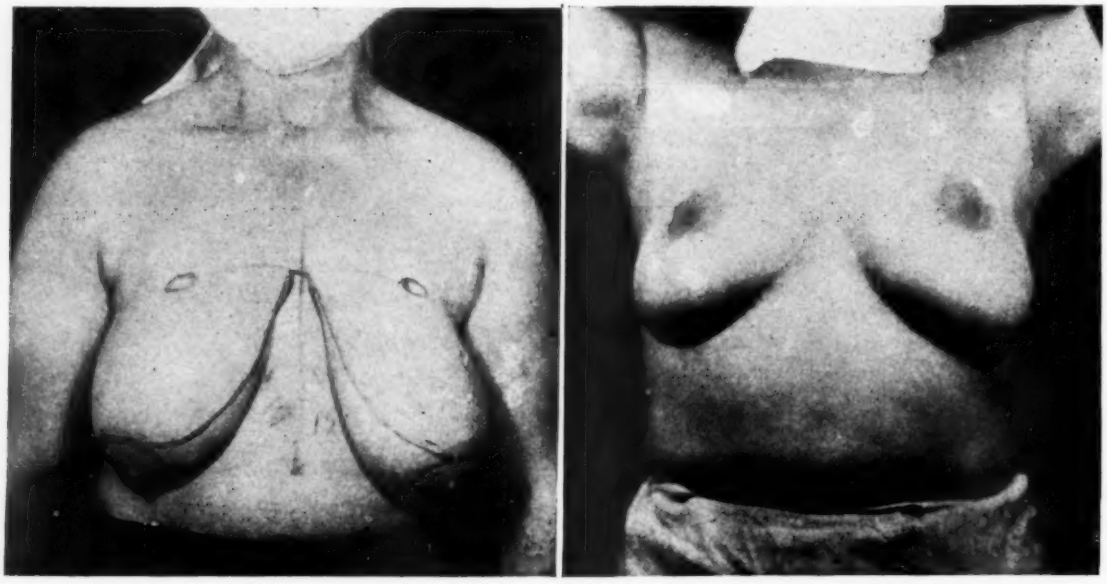
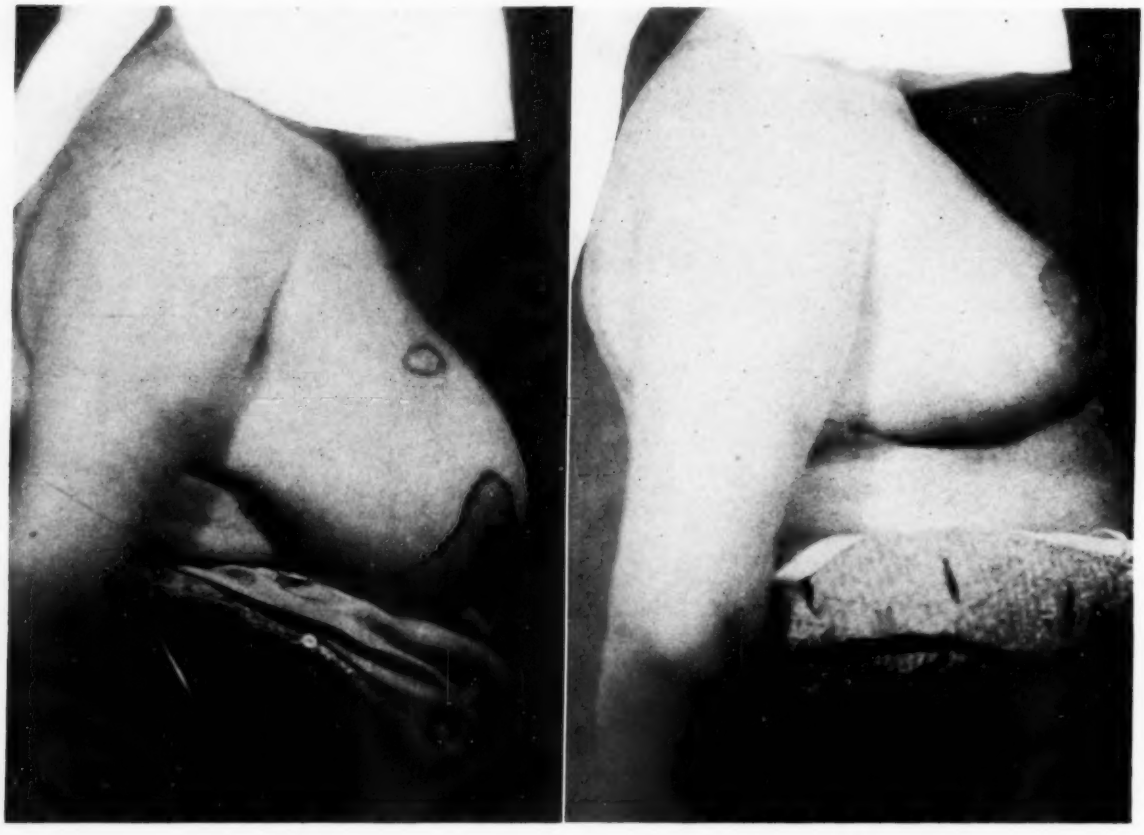


Fig. 10. Side view showing all sutured parts fully finished.



A B

Fig. 11. A, hypertrophy of the breasts in a woman 45 years old, with the arms in normal position; B, the result of the operation and suture lines, with the arms in a raised position, two months after removing the stitches. Note absence of scars.



A B

Fig. 12. A, mammary ptosis in an opera singer, 39 years old, mother of four children B, the result twenty days after removing the stitches.

together, being careful to provide a small drain at the outer third of this line, which I generally remove after three days (Figs. 7 and 8).

The entire operating field is then covered with cicatul or tulle grase lumiere. After having been properly bandaged the patient can return to her home, it being advisable for her to keep relatively at rest for two or three days.

The sutured stitches of the areola and the submammary fold which follows line B-C are withdrawn on the tenth day to the extent of half of their number, and the remaining half are removed on the eleventh or twelfth day (Figs. 9 and 10).

ADVANTAGES AND DISADVANTAGES

Among the advantages I consider the following:

1. General anesthesia is not required.
2. The patient is able to go home shortly after the operation.
3. The entire operation requires but one sitting, meaning that a minimum of time is wasted, which may be of great economic importance to people who are employed.
4. The gland remains solidly fixed in its new position in the exact size desired.
5. The danger of necrosis of the areola and nipple, which unfortunately is so frequent in other procedures of operation, is eliminated.

Among the drawbacks to this method I shall mention the following:

1. The total time required for the operation is longer, sometimes about three hours or more being necessary.
2. The surgical treatment by this method must be exercised with a larger amount of skill rather than the usual procedures.

SUMMARY

Having thus far obtained satisfactory results with this technique for the surgical treatment of pendulous breasts, its description is presented in detail.

INGROWN TOE-NAIL*

A CLINICAL STUDY

CARL J. HEIFETZ, M.D.

Surgeon of the Staff, Jewish Hospital of St. Louis

ST. LOUIS, MISSOURI

IN spite of all that has been written on ingrown toe-nail in the past, one must admit that (1) there is no concise and generally accepted method of treatment, (2) in the hands of most surgeons practically all methods of treatment still result in occasional recurrences, (3) conservative methods of treatment require too much time and are frequently ineffective, and (4) the incapacitation from surgical treatment is entirely out of proportion to the severity of the lesion.

Our purpose is to summarize the history and present status of the subject, and to present a routine of treatment which incurs a minimum loss of time to the patient and which the writer has used for several years with great success.

"Ingrown toe-nail" is a misnomer. It is generally conceded that the underlying pathology is not the growth of the nail into the flesh, but rather the growth of the flesh upon the edge of the nail.[10,18,51,60,86] More descriptive are the terms "infleshed toe-nail"[13,21] and "embedded toe-nail,"[3] but the common usage of the first name has given it a status of approval similar to that of other medical misnomers.

ANATOMY AND PHYSIOLOGY OF THE NAIL

The nail is implanted into the skin proximally by the *root*, the exposed portion being called the *body* and its distal end the *free border*. The two lateral borders are overhung by margins of normal skin, the *nail-walls*. Proximally overhanging the root is a thin parchment membrane, the *eponychium*, or cuticle, which is a distal extension of the *stratum corneum* of the skin. The nail-plate itself is a specialized horny layer of stratum corneum. Proximally near the root a white, opaque, semilunar area, the *lunula*, can be seen. It is sometimes necessary to push the eponychium back a bit to get a good view of this. The nail plate lies on a fibrous, white, cartilage-like layer known as the *nail-bed*, a continuation of the *stratum germinativum* of the adjacent skin. The portion of the nail-bed underlying the lunula is called the *matrix*. This structure consists of a half dozen layers of cells from the stratum germinativum, having a brownish tint but appearing white in reflected light and imparting the normal white color to the lunula. These cells gradually pass over into the root of the nail.[28,56,67] (Fig. 1.)

The matrix is of considerable importance in this discussion, as it alone is concerned with growth of the nail.[56,67] This was clearly demonstrated by Quénu in 1887,[72] and has been confirmed by other observers.[22,60] However, one occasionally reads statements that the nail grows also from the "fold of eponychium overlying the nail and its junction with the cells proximal to the nail,"[83] and from the nail-bed.[28] These statements show lack of careful observation. It is true that if the nail-plate is removed and the matrix destroyed, the nail-bed will secrete a thin, lamellated, cornified, epidermoid layer, which has been mistaken for regenerated nail.[60,72] However, this lacks the texture, the rigidity and the structure of the nail-plate. Another important, almost axiomatic observation is that every part of the nail-plate grows straight out from the corresponding part of its root, e.g., the medial side of the root will

* From the Surgical Service of the Aaron Waldheim Health Clinic, Jewish Hospital of St. Louis.

eventually grow out to form the medial side of the free border.

The writer has been able to demonstrate repeatedly these principles of nail growth during his clinical observations.

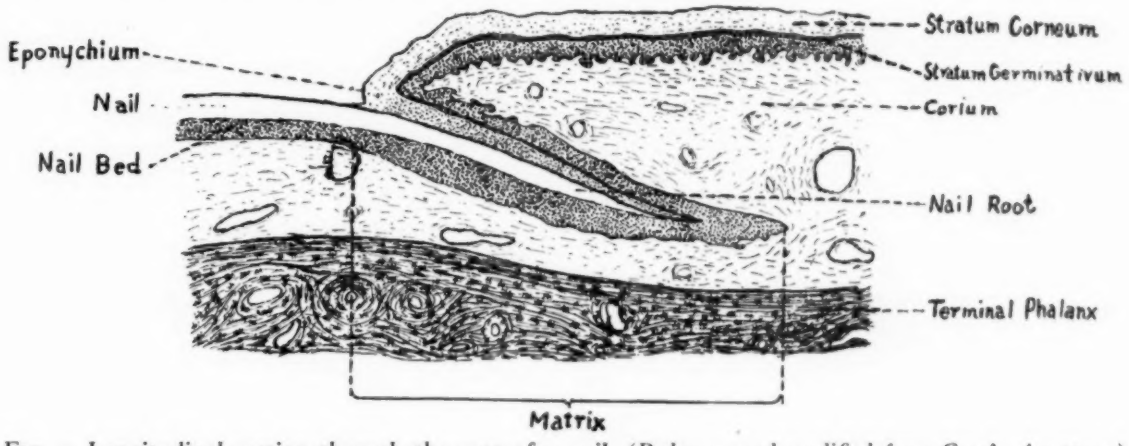


Fig. 1. Longitudinal section through the root of a nail. (*Redrawn and modified from Gray's Anatomy.*)

ETIOLOGY

Ingrown toe-nail is a relatively common lesion. Dardignac,[15] who had under his care 6000 infantry men for a period of seven years, observed that 2 per cent developed this affection at one time or another.

Nearly all authorities agree that the condition is most prevalent amongst young adults.[8,10,40,60] Gosselin, quoted by Block,[8] reported forty-one of fifty-four cases under twenty-one years of age. My own cases are classified according to age as follows:

Years	No. Cases
12 to 14	5
15 to 19	9
20 to 24	8
25 to 29	2
30 and over	8
Total cases	32

The youngest patient was 12 and the oldest 73 years. The explanation for this predominance among younger people is not entirely clear although the amount of walking may play a role. It is possible that the rapid increase in size of the foot and nail occurring in adolescence is not accommodated for by a frequent change in size of the shoes.[8] The result is an increase of pressure on the toes, which, as will be shown later, is largely responsible for the development of the condition.

In the middle of the last century Gosselin reported a preponderance of males to females of 5½ to 1, and Bouchaud of 2½ to 1.[10] Nicaise[60] also reported a greater number in men. Recently there seems to be a relative increase of the number of cases in females, so that Keyes[40] reported three-fourths of them in females, and in my cases there were 15 males and 17 females. It is possible that changes in shoe styles have been a factor in this relative increase in females. Women's shoes have certainly become narrower at the toes and higher heeled, tending to increase the pressure on the toes.

There has been no evidence to show that occupation predisposes to this condition. The amount of walking done by the individual, however, has been blamed by some investigators.[8,15,24] In favor of this is the fact that, in spite of more comfortable and roomy shoes, the condition is usually considered as frequent in males as in females, and there is no question of the greater amount of walking done by men. Also, military surgeons in charge of large numbers of infantry have found this to be one of their most troublesome minor surgical problems. On the other hand, Bouchaud[10] expressed the belief that people who do a large amount of walking have

developed a sufficiently hardened skin to resist the development of this condition.

Several other factors have been cited as underlying etiological factors. Gross,[29] Hickman[30] and Morris, quoted by Block,[8] having seen several cases in individuals of the same family, assumed there was an hereditary predisposition to the lesion. It is more likely that these families were prone to purchase their ill-fitting shoes in the same place. Nicaise[60] described a type of great toe, flattened, or spatula-like, which he believed when present predisposed to this affection. However, he was careful to note that it may also develop on a previously normal looking toe. It hardly seems likely that direct injury can be considered a causative agent, although Nicholson[61] has described the condition following a blow to the toe. No other investigator has stressed this point. Certain it is that direct trauma to the toe is very common, much more common than the occasional development of ingrown toe-nail would lead one to believe.

Ingrown nail occurs almost exclusively on the great toe of either foot. The fact that it never occurs on the hands, and almost never on any of the other toes, indicates that there must be some conformation of the great toe and nail that predisposes to its development. A glance at the normal great toe, comparing it with the adjacent toes, will reveal a real difference. The greatest width of all the nails except the great toe is only slightly less than the width of the digit itself, thereby leaving very little wall of soft parts on either side to overhang the nail edge. However, in the average great toe, the greatest width of the nail may occupy only two-thirds the width of the digit at that place, leaving a good margin of nail-wall on either side to overhang the nail edge.[6] I am skeptical if any case of ingrown toe-nail ever develops on a toe other than the great toe, notwithstanding several reports to the contrary.[8,15] I also have seen two individuals who presented lesions resembling ingrown toe-nails on digits other than the great toe, one having two toes so involved. Examination revealed that the nail itself was not embedded, but that there was a chronic draining infection of the nail-wall, accompanied by swelling and pain and having the characteristics of a fungus infection. In both cases fungicide treatment was instituted; in one the edge of the nail was packed away; in the other it was not. Both cleared up in about the same period of time. In one case, the nail was so loose that it was easily lifted off its bed. I have not seen a similar loosening by infection of the nail of the great toe, but there is no reason why it might not occur in fungus infections of that toe.

Most writers have found the condition more common on the right toe than on the left.[8,15,40,80] My figures show the left a bit more frequently involved: eleven were on the right, fourteen on the left, and seven on both feet. Stewart[80] has made the interesting observation that the right foot is more commonly involved in right handed individuals, while the left foot seems more frequently involved in left handed persons. For practical purposes one must conclude that the right foot is slightly more frequently involved than the left, and that both feet are affected in about one-third to one-fourth of the cases.

There seems to be some dispute as to which border is more often affected. To eliminate some of the confusion sometimes encountered in the literature, we shall designate that edge of the nail adjacent to the second digit as the peroneal border and the other border the tibial border. Early writers[18,60] were of the opinion that the peroneal edge was more commonly involved. Ney[59] found the condition more frequent along the tibial border. My own thirty-two cases show involvement of twenty-six peroneal and twenty tibial borders.

The immediate causes of ingrown toe-nail are pressure due to foot-wear and improper cutting of the nails. Nearly

all authorities agree that ill-fitting footwear forms the basis of most of the trouble.[3,6,8,15,26,59,60] When one considers that ingrown toe-nail is a disease of civilization, that savages and other people who habitually go barefooted rarely develop it,[10] and that the condition will generally clear up if a patient is bedridden because of a prolonged illness, there is little room for argument on the role played by footwear.* The shoes act as a wedge in which the toes are cramped into a smaller space than their normal spread requires. The result is pressure on all the toes, but probably more on the great toe because of its function in locomotion, its greater prominence, and because most faulty shoes present an outcurving of the medial border of the last which exerts a direct pressure on the tibial edge of the great toe. (Fig. 2.) A similar condition will result from the wearing of high heeled shoes. In this case the foot slides forward and the toes are cramped in the conical shaped front part of the shoe. Although stockings conform more easily to the shape of the foot, it is conceivable that they too may exert a confining effect.[26]

One can readily visualize how this pressure acts on the tibial edge of the toe. Because the great toe has a wide overhanging nail-wall, the edge of the nail acts as a fixed dull cutting edge, over which the soft parts are squeezed every time the patient takes a step. The pressure exerted on the peroneal nail edge is less direct. Here also there is a thick nail-wall, but it is not the direct pressure of the shoe, but rather that of the second digit that is responsible for the development of the lesion. If one

Fig. 2. Diagram showing the direction of the pressure exerted by the outcurving medial border of an incorrect shoe against the tibial border of the great toe.

examines the toes as they lie in the formation of a peroneal edge lesion, it is usually possible to demonstrate that the second digit lies somewhat below the plane of the great toe. When the foot is then confined by a tight shoe, the soft parts on the peroneal border of the great toe are pushed up over the peroneal nail edge,[8,10,15,31,41,60] every step of course aggravating the condition.

Improper cutting of the toe-nails is almost universal. Most people trim their toe-nails as they do their fingernails, except that the trimming is usually done more closely. Particularly is this true of the great toe-nail, as this nail has the tendency to bore holes through the ends of closely fitting stockings. When the trimming is carried closely around the edges, a sharper and more deeply embedded edge is left be-

* Recently my attention has been drawn to two cases of "ingrown toe-nail" developing in patients who were chronically invalided and almost completely bedridden. One of these patients had multiple sclerosis, the other pulmonary tuberculosis. Both of these cases had abnormal nail curvatures resulting from trophic changes such as ridging, thinning, malformation and brittleness. The nail edges exerted what amounted to a cutting action on the nail-walls. The findings usually seen in ingrown toe-nails, as thickened nail-wall, rigid nail-plate and relative narrowness of the nail, were absent. Added factors of pressure of the bed sheets and inadequate circulation (both patients had absent dorsalis pedis pulsations) might suffice to complete the etiological picture. It is likely that except for such trophic changes, any increased convexity of the nail that might be present is the result of increased pressure, rather than the prime cause of ingrown toe-nail.

hind,[10,26,30,51] the better to irritate the adjacent soft parts, especially when tightly fitting shoes are worn. Once this irritation has begun, the patient, believing the nail to be the aggressor, more closely trims away the offending portion. Since he can trim it back only part of the way, the remainder being deeply embedded, he leaves behind a small sharp spur, which further aggravates the condition by jutting sharply into the soft parts. (Fig. 3.)

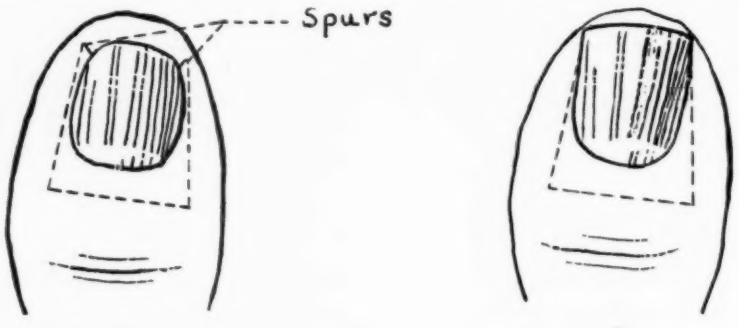


FIG. 3. A. Improper method of cutting the toenail. Embedded spurs remain. B. Proper method of cutting the toenail.

SYMPTOMS AND COURSE

Once the lesion has begun it takes but a short period of neglect to produce the usual symptomotology associated with this condition. For the sake of convenience in treatment, the course of the illness can be divided into three stages.

In the first stage the patient begins to feel pain along the edge of the nail, which increases when he walks around. The only noticeable findings are a redness and slight swelling of the soft parts bordering the lateral margin of the nail near its free border. When this area is pressed downward, i.e., away from the nail margin, little or no pain results; but when it is pushed against the nail-edge, the pain of which he complains when he walks is duplicated. In most cases this will and does subside because the patient diminishes his amount of walking, or he changes to another shoe, or even because he goes to bed for his usual night's sleep. In fact, most cases will subside if the patient just leaves the nail alone. Sometimes he does trim away the mechanically offending portion without leaving a spur behind, and this relieves the condition.

If the lesion progresses, however, it passes to the second stage. This stage may develop comparatively early in the course of the illness, after several weeks or months, or even after the first stage has developed and retrogressed several times previously, all of course depending upon the number and degree of etiological agents which are allowed to remain. One then notices a gradual change to a more marked swelling of the soft parts, so that there is a definite lapping of the soft parts over the nail-edge. The edge of the nail then causes a minute abrasion, which is not permitted to heal because of the constant irritation by the nail-edge whenever the patient walks. At first a thin sticky secretion is present, but in a location so plentifully supplied with microorganisms infection rapidly occurs and the discharge then becomes more profuse, thicker, cloudy and fetid. If the hypertrophic nail-wall can be retracted sufficiently to visualize the depth of the sulcus, one notices that the abrasion has developed into a grayish ulcer covered by exudate and surrounded by an inflammatory, reddened, thickened and often discolored area. In this stage the slightest pressure of the soft parts against the nail-edge causes marked pain. Walking is at best difficult, often impossible. Seldom does the condition retrogress spontaneously, and then only after the patient himself relieves the toe of all pressure by refraining from walking or by walking with a cut-out shoe,

or by the good fortune of cutting the nail-edge away without leaving a spur. Even after an apparent cure, the condition will frequently recur as soon as the patient resumes his regular activities.

The second stage will generally continue to the third stage if enough time is permitted to elapse. In this stage, as a result of continued irritation of the ulcer by the nail-edge, a chronic inflammatory reaction is set up, characterized by the formation of granulation tissue arising in the ulcer and extending along the sulcus between the nail and the nail-wall. These granulations soon become exuberant, bleed readily, and increase the already great swelling of the nail-wall. They embed the nail-edge by completely overhanging it. (Fig. 4.) The surrounding soft parts are usually purplish and are extremely tender. After a prolonged period of suffering the patient develops either a great tolerance to his pain or the nerve endings are partly desensitized, so that one is occasionally surprised to see a patient who has walked around for several weeks during this stage. Once granulations have developed, it is questionable whether a lasting cure ever occurs spontaneously.

TREATMENT

Most authors have fairly uniformly agreed on the matters of etiology and symptoms of ingrown toe-nail, or have been at only slight variance from the commonly accepted views, but it is in the matter of treatment that there has been such uniform disagreement. It is obvious that when one condition has been productive of so many varieties of treatment, probably none has been entirely satisfactory. Each writer of course believes his own method to be the best, and probably it is, in his own hands. A brief classification and review of the various methods of treatment advocated are of distinct interest. The methods can be best grouped as follows:

1. Prophylactic measures.
2. Conservative (non-operative) measures.
3. Quasi-conservative measures.
4. Radical operative measures.

Prophylactic Treatment. The best time to cure any disease is before it begins. The incidence of ingrown toe-nail could certainly be reduced by scrupulous prophylactic measures,[15,26,50,78,86] but as long as shoes are worn, many cases will continue to develop. Proper shoes are readily available but rarely worn. It is not the purpose of the writer to describe adequate footwear, as numerous descriptions are given in orthopedic textbooks. Suffice it to say that women rarely and men seldom wear such footwear. The utilization of proper footwear would seem to have its value, so far as prophylaxis is concerned, only in those cases that have already developed one ingrown toe-nail and will submit to correct shoes to prevent a similar disorder on the other toe or nail-edge.

Fig. 4. Photograph of an advanced case of ingrown toenails in which there is a great overpiling of granulation tissue on the peroneal borders of both great toes. So much of the nails is covered that they appear to have been cut.

However, even with the use of improper footwear, correct trimming of the nails would likewise greatly diminish the incidence of ingrown toe-nail. Figure 3B illustrates the correct method of trimming toe-nails. It should be observed that the nails should be allowed to grow to a con-

siderable length and then cut straight across. This accomplishes two things: (1) the corners become sufficiently distal so that they are unable to press into the soft parts; and (2) if the nail is sufficiently long, the pressure of the shoe on the convex center of the end of the nail will tend to push the corners upward, away from the soft parts. There are two possible objections to allowing the nails to grow in this manner: (1) it is easy to perforate the ends of the hose, which can be largely prevented by wearing slightly oversized hose; and (2) occasionally the sharp pointed corner of the nail will stick into the tissue of the adjacent toe. This can be remedied by cutting away a tiny portion of the corner of the nail by a small diagonal cut.

Conservative (Non-operative) Treatment. This includes (1) separation of the nail from the soft parts, (2) treatment of the soft parts, (3) bandaging of parts in position, and (4) mechanical appliances.

The first method has been and is that most commonly used. This form of treatment has as its basis the separation of the ingrown toe-nail from the soft parts by the interposition of some foreign material. In 1619, Fabricius Ab Aqua Pendente described a method of inserting a small piece of lint dipped in warm water between the nail edge and the soft parts, which he replaced from time to time (quoted by Loewe[48]). Undoubtedly this method had been used for many centuries. Modifications of this have been repeatedly advocated. When cotton became more common than lint it too was used.[6,26,35,50,70] Later the pledgets of cotton or lint were dipped in various solutions, mostly astringent, such as persulphate of iron,[9] glycerine,[36] "black mercury lotion,"[36] collodion,[35,54] silver nitrate,[26] etc. Other materials were likewise interposed, e.g., tin plate (Desault quoted by Wardrop[86]), lead plate (Boyer quoted by Hunter[35]), cork,[20] silver plate.[42] One author[35] described a small spatula-like instrument which rendered insertion of the pledgets easier and less painful. It was also claimed that insertion became easier if a longitudinal groove was cut almost completely through the nail near the involved margin, since this made lifting of the ingrown edge much easier.[3,7]

Even today this method or one of its modifications remains a most satisfactory mode of treatment, with this limitation, that it is often impossible to insert anything between the nail-edge and the adjacent inflamed tissue, because the pain is too great or the corner of the nail has been cut too closely.

Some authors, asserting the soft parts are to blame, described treatment designed to reduce the hypertrophied tissue. For this, a silver nitrate stick was most commonly advocated,[3,7,36,47,86,88] either alone or in conjunction with removal of some of the nail.[7,36,58,88] Frequent applications of copper or zinc sulphate were claimed to have a similar action.[47] Hot wax was poured into the ulcer or granulations by Stafford (quoted by Leonard[47]). Alum,[68] "liq. potassi,"[20] tannic acid,[53] and tincture of ferric chloride[34] had their advocates.

Those writers who held that the malposition of the second digit was responsible for the development of the ingrown toe-nail on the peroneal nail-edge treated their cases by bandaging the first and second digits together in such a way as to force the second digit to lie on the soft parts instead of under them.[41] Others placed a small pledget of cotton on the soft parts, pushing them away from the nail-edges, in the hope that this would effect a cure in the ambulatory patient. Nicholson[62] used a mold of gutta percha or flexible lead which he fitted tightly and secured over the dorsum of the toe. Bouchaud[10] devised a semicylinder of zinc under which two sections of cork were glued to fit into the sulci between the nail and the soft parts. The resiliency of the cork exerted a pressure downwards on the soft parts when the patient walked. Webb[87] described the use of silver wire molded and fixed in such a way as to lift up the nail. A modification of this was recommended by Foote, quoted by Graham.[26] The obvious objection to all of

these mechanical devices is their difficulty in application and maintenance. Of course, many times they do not effect a cure.

All conservative measures have the limitation that they are often not curative but only palliative. It can be said that once granulations have developed (stage three), few conservative measures will effect a lasting cure. Temporary relief may be established but that is all. In the second stage (drainage without granulation), cures may be obtained but frequently recurrence will occur at a later date and more radical measures will be necessary. It is only during the first stage of the illness that conservative measures will effect a lasting cure, provided that the prophylactic measures previously mentioned are likewise carried out. In this case none of the other measures seem to have any advantage over the time-honored and simple method of inserting a pledget of cotton between the nail and the soft parts. Even in those cases where a cure is effected, the time element is an important factor. All these measures imply a gradual and slow relative change in the position between the nail-edge and the soft parts. This seldom takes less than three weeks and often more than eight weeks. The pain resulting from the application of the several treatments is frequently very severe. It can therefore be seen that the type of treatment is anything but satisfactory in the majority of the patients.

Quasi-conservative Treatment. In view of the unsatisfactory results obtained by these measures, minor operative procedures were directed to the nail or the soft parts, which measures we have preferred to term quasi-conservative. The simplest of these procedures is frequently done by the patient himself, who excises the corner of the nail. A great many writers[14,23,25,29,36,47,55,75,81,86] advocated the removal of a strip of nail from 1/16 to 3/8 inch wide on the involved side, tearing it away from its nail-bed, usually without any anesthesia. Some surgeons[45,86,88] excised a wedge or v-shaped portion of the nail from the center, boring holes on each side and pulling the edges of the v together with wire inserted through these holes. Others[26,62] scraped the center of the nail thin with a piece of glass or scalpel, with the idea of making the edges easier to elevate. In the early 1800's the method of Dupuytren consisting of the evulsion of whole or half of the nail without anesthesia was the most commonly advocated procedure.[5,36,58,75,79] The brutality of this procedure is manifest when one considers that this was a form of torture used by savages on their captives. A more logical method of complete removal of the nail was its dissolution by application of chemicals such as caustic potash,[2,36,38,53,71,88] silver nitrate—linseed meal poultice,[49] or glacial acetic acid.[12] Although the methods of treatment directed to the nail were for the most part temporarily satisfactory, recurrence was so frequent when the nail resumed its growth, that these methods have been largely discarded today. Since a nail takes on the average six months to regrow, many of these cases were considered cured soon after their treatment, whereas observation at a later date would reveal a high percentage of recurrences.

Several operative procedures on the soft parts, some of considerable formidability, were described. The earliest procedures of this type dated back to Paul of Regina (A.D. 668), Abul Kasem (1106) and Ambrose Paré (1508), who recommended excision of the soft parts embedding the nail and cauterization of the wound.[88] Gradually more of the soft parts were excised[11,19,21,37,63,76] until Cotting[13] recommended excision of all the diseased tissue and a large margin of healthy tissue en masse, baring the entire nail-edge, healing taking place by granulation and subsequent cicatrical contraction.[39] This method was further modified so as to reduce the time of healing by covering the operative wound on the second day with a Thiersch graft obtained from the arm.[84] Another operation on the soft parts was the excision of a wedge of soft tissues parallel to the nail-edge and suturing

the wound edges together, thereby reducing their bulk and pulling them under the nail.[18,33] Still another was the somewhat complex method of Ney,[59] using the pedicle flaps to cover a wound resulting from the excision of the soft parts adjacent to the nail. All these operative procedures on the soft parts, although frequently resulting in satisfactory cures, have been largely discarded for one or more of the following reasons: (1) The time of healing (from three to six weeks) was so great as to render them impractical. (2) There was a high percentage of recurrences. (3) The resulting scar was frequently tender and annoying. (4) Bleeding was often difficult to control. (5) The use of skin grafts required two operative procedures instead of one. (6) The use of sutures in such a contaminated and sometimes infected field was contrary to established surgical dicta.

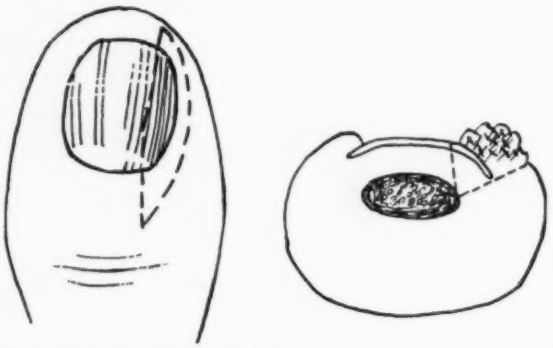


FIG. 5. The elliptical wedge operation for ingrown toenail.

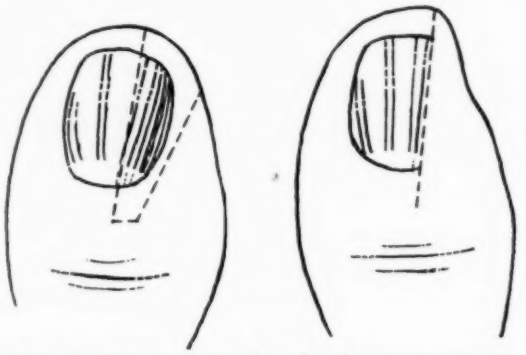


FIG. 6. Anger's operation for ingrown toenail.

Various combinations, too numerous to mention, of two or more of these methods have been described from time to time.[4,30,52,74,77] The most frequent was the combined excision of a margin of nail and adjacent involved soft parts.[24,27,31,36,66,80]

Radical Operative Treatment. It has been previously shown that the soft parts are the site of most of the pathology; but treatment directed to the soft parts has frequently proved unsatisfactory. On the other hand, it is conceded that if the nail were not present this pathology could not occur. It is therefore obvious that if the nail were prevented from regrowing after it were once removed, it would be impossible for the condition to develop again. This is the basis of the so-called radical operations for ingrown toe-nails. There is only one method to prevent regrowth of the nail, namely the destruction of the matrix from which it grows. All radical procedures, though some are less formidable than many of the quasi-conservative measures, aim at excision or destruction of all or part of the matrix.

The earliest method of this type, complete excision of the matrix through a semilunar incision, was described by Dupuytren (quoted by Robbe[75]), but was completely ignored subsequently because of lack of satisfactory anesthesia. The advent of general and local anesthesia soon resulted in many radical operative procedures. That most commonly used today consists of an ellipitical wedge excision of the nail-edge together with the corresponding portion of the matrix and the adjacent soft parts, all in one piece.[16,26,32,43,48,50,60,65] (Fig. 5). There are several modifications of this: (1) excision of nail and soft parts, followed by daily applications of carbolic acid to the matrix;[69] (2) preservation of the nail-wall in the form of a lateral flap, while excising the matrix and nail-edge;[22,44,82,85] (3) the method of Anger,[1] so popular at the turn of the century, consisting of block excision of all the terminal phalanx on the side of the bone except for a lateral skin flap (Fig. 6), later modified by applying sutures to hold the flap in place.[17,39]

Many operations were aimed at complete destruction of the matrix, irrespective of whether the cases were uni- or bilateral. Some of these were the complete excision of the matrix together with the overlying eponychium en bloc, in the form of a semicircular wedge[57] or in the form of a quarter-moon;[75] excision of the matrix in the form of a square by turning back an eponychial flap.[15,72] Others extirpated the nail and cauterized the matrix with heat[35] or with nitric acid.[64] Still another[83] cut out a deep groove extending down to the bone on all three circumscribed borders of the nail. The most radical procedure was the amputation of the end of the terminal phalanx together with all the nail, matrix and nail-bed, using different types of flaps to effect closure.[34,46,78,90]

It is not our purpose here to discuss the relative or collective merits of these various operations. Undoubtedly, they all give a reasonable hope for a permanent and effective cure. Most methods commonly used today result in some recurrences[40] but their number has been so greatly reduced that it is generally conceded that radical treatment of one form or another is the procedure of choice in advanced cases. The subject then resolves itself to: what are the objections to radical procedures? and what methods largely obviate these objections?

Any of the radical measures mentioned above are objectionable on one or more of the following grounds: (1) The disfigurement to the toe is too great; this is particularly true of the methods of amputation and some of the more complete matrix excisions. (2) The healing time is too prolonged; all methods requiring healing by granulation, and the most commonly used method (Fig. 5) have this objection. To remedy this situation, the use of a small Thiersch graft applied to the defect has been recommended.[73,82] This procedure raises the next objection. (3) The method is unnecessarily complex. (4) The use of sutures in several operations to effect primary union of the flaps often defeats its own purpose, as the field is greatly contaminated. (5) The accompanying pain is too great. (6) All procedures require

Fig. 7. Photograph of an island of nail regrown from inadequately excised matrix. The nail border and island have been marked with ink.

that the patient be non-ambulatory for a period of three to fourteen days.

There seems to be one method of radical treatment, that of Winograd,[89] really a slight variation of that described earlier by Foote,[22] which when properly performed, with a few minor variations, seems to obviate all these objections. This procedure, described later in more detail, has now been adopted by the author in all cases requiring radical treatment. The results in the present series of twenty operative cases seem fully to justify the faith in the procedure. Prior to this, several operations were attempted adhering to the technique described by Winograd, and, although the objections mentioned were largely obviated, the resulting recurrences and regrowth of islands of nail from incompletely removed matrix (Fig. 7) made it necessary to work out the details of the procedure more scrupulously.

RECOMMENDED TREATMENT

The writer has adopted the following procedures in caring for ingrown toe-nails, depending upon the stage in which they are seen:

First Stage (early inflammation of the nail-wall). These are all treated conservatively. If the nail corner is of sufficient length, a small pledget of cotton moistened with alcohol is inserted gently between the edge of the nail and the soft parts, in such a way as to allow some of the cotton to extend slightly under and slightly above the nail edge. This is best done by pulling the soft parts away from the nail, so as to expose the edge, and inserting a thin wick of cotton with the flat end of a small probe. The excess alcohol is allowed to dry or is blotted off with dry cotton, and a small amount of collodion is applied to the inserted cotton and allowed to dry. This seems to hold the cotton in place. The patient is now reminded that cleanliness of the feet is necessary, and is instructed as to the advisability of obtaining proper footwear, both to aid in the present treatment and to prevent any future similar condition. He is also instructed in the proper method of trimming toe-nails. Frequently he is unable or does not care to buy new footwear. Or he will ask if he can wear a house slipper or an old shoe during treatment, which I gladly allow him to do, since in this stage it is hard to obtain any concessions from the patient. The patient returns in a week, or in less time if it is feared that a pressure sore will result. Sometimes the condition is completely relieved, making it unnecessary to repeat the treatment. In most cases, however, it will be necessary to reinsert pledgets of cotton at weekly intervals until the corner of the nail has grown sufficiently to make its embedding improbable.

Sometimes, when the patient is first seen, the corner is so closely trimmed that insertion and retention of the cotton is impossible or too painful. The patient is then instructed to soak the foot in water slightly above body temperature for an hour, once or twice daily. He is permitted to be ambulatory, but he must wear a house slipper or a cut-out shoe. He is then told to return twice a week, each time to see whether the corner has grown sufficiently to insert cotton. From this point the procedure is exactly as previously described.

Of thirty-two patients, six were seen in the first stage, four being treated by inserting cotton on their first visit, the average cure taking three weeks. Two had cotton inserted a week later, the cure requiring four weeks in one and six weeks in the other. Five patients have been observed for at least six months without evidence of recurrence. Track of one patient was lost after six weeks, at which time there was an apparent cure.

Second Stage (period of drainage). When a patient presents himself in this stage, choice of treatment rests largely with him. An attempt is made to explain the following points to him. (1) It is not possible to promise lasting cure by conservative measures. (2) Even if relief is obtained by these measures, the chance of recurrence is quite great. (3) The amount of pain endured during the period of conservative treatment is in the aggregate probably greater than that resulting from operative measures. (4) A lasting cure will result from operative treatment. (5) Operative treatment requires the loss of only one day from work, the day of operation. (6) There is very little pain attendant upon the operation, and the only real inconvenience is the wearing of a cut-out shoe for one to two weeks. If these probabilities are balanced by the patient, he will generally choose the operative treatment.

A total of fourteen of thirty-two patients presented themselves during the second stage; ten received operative treatment, four conservative treatment. Of these four, one patient required eight weeks for complete relief, but has recently returned after eleven months with an early recurrence, which he still desires to have treated con-

servatively. One has purposely been treated conservatively because of his advanced age, 72 years, and poor circulation in the extremities (arteriosclerosis); the treatment took five weeks, and he is free of symptoms after eight months. The other two are also free of symptoms after prolonged periods of treatment. On the whole, these patients were more difficult to manage, seemed to complain of more pain, and were on the average more incapacitated than the patients who had undergone operation.

When a patient presents himself during this stage and the nail is so closely trimmed that immediate conservative treatment is not possible, operation is unequivocally recommended.

Third Stage (period of granulations). Operative treatment is recommended to all these patients. They have usually suffered so much pain that they gladly welcome it. There is little doubt that many patients in this stage have been cured by less radical or even non-operative measures, but usually at the expense of more suffering than the operation entails. Ten patients, all of whom were operated, presented themselves in this stage.

OPERATIVE PROCEDURE

Preoperative. For three to five days before operation, depending upon the degree of infection present, the patient is instructed to soak and cleanse his foot in a bath of warm water (110°F.) for an hour twice a day. White socks are preferable and are to be changed daily. If walking is difficult, a cut-out shoe should be worn. A clean sock and an old shoe should be brought along to the operation.

The inflammatory condition of the soft parts does not require postponing the operation. Only a frankly acute suppurative process would warrant postponement of the procedure.

No premedication is necessary.

The foot is prepared with iodine and alcohol and draped. Care should be taken to cleanse thoroughly with an applicator of iodine the lateral and subungual sulci.

Since the success of the operation largely depends on the adequate exposure and visualization of the matrix, it is necessary to render the field bloodless by the application of a tourniquet. Several turns of an ordinary elastic rubber band are applied around the base of the toe and pulled up snugly with an artery forceps. The tourniquet is applied immediately before injecting the local anesthetic, because it seems to render injection less painful and it confines the anesthesia to the region desired.

Anesthesia. Local anesthesia is certainly the procedure of choice. The only reason for using general anesthesia in any form would be at the insistence of an extremely apprehensive and nervous patient, or in an uncooperative child. However, the condition is extremely rare in the preadolescent child. The advantage of nerve block over local infiltration is manifest, since it avoids any injection into an already infected field. Nerve block anesthesia when properly applied is always successful. There are four digital nerves of the great toe, two dorsal and two plantar. The dorsal digital nerves, terminal branches of the superficial peroneal nerve, supply the skin on either side of the dorsal surface of the toe except the structures of and around the nail. The plantars are branches of the medial plantar nerve and supply the skin on either side of the plantar surface of the toe. Upon reaching the terminal phalanx, they send twigs around to the dorsum to supply the structures of and around the nail.[28] Strictly speaking, then, it is necessary to block only the plantar nerves. However, they run in so close approximation to the dorsal nerves on each side of the first phalanx, that any injection of the plantars will generally block the dorsal nerves. The injection is made through a three-fourths inch hypodermic needle, using 2 per cent procain hydrochloride, without epinephrine. The needle is thrust suddenly through the skin on the side of the toe, about one-fourth inch distal to the

tourniquet, an attempt being made to strike the periosteum of the proximal phalanx on the first thrust. The needle is

Fig. 8. Incision (inset) and dissection of flaps to expose root of nail.

then withdrawn about an eighth or quarter inch, and 2 to 3 c.c. of the anesthetic solution injected. The direction of the needle is then changed a bit plantarward and then dorsally, in which places 0.5 to 1.0 c.c. of the solution is further injected. A similar procedure on the other side of the toe will complete the anesthesia and the operation can be started in five to ten minutes. Injection of one side of the toe alone has not given satisfactory anesthesia for operation on that side.

Steps of the Operation. 1. An incision three-eighths inch long is made in the eponychium and proximal nail-wall, extending slightly diagonally laterally from a point on the nail corresponding to the line on which the nail will presently be excised. This is made deep enough to strike the root of the nail. Medial and lateral flaps are dissected along this line so as to expose at least the lateral third of the nail-root on that side. The lateral flap should also include sufficient tissue so as to expose the embedded nail edge. (Fig. 8.)

2. A small thin flat spatula similar to the Bollenger-Hajek nasal elevator (Fig. 9) is then inserted beneath the free border to the nail between the nail-plate and the nail-bed along the line where the nail is to be cut, and, hugging the undersurface of the nail, is pushed proximally until it emerges

Fig. 9. Ballenger-Hajek nasal elevator used in separating nail from nail bed.

in the incision proximal to the root of the nail. This spatula is then worked laterally until it lifts the involved portion of the nail from the nail-bed. This procedure takes but a second or two, and makes it unnecessary to tear the nail out after it is cut, which procedure sometimes leaves the proximal corner behind.

3. Using a small scissors, the freed involved portion of the nail is excised along a straight line, exposing the nail-bed and, proximally, the matrix on that side. About a third of the nail should be removed, but this can be varied as befits the case.

4. This step is the most important of the operation, and extreme care should be taken to perform it properly. Using a sharp small bone curette, the exposed matrix is completely curetted away. It is necessary

to remove every bit of this matrix, since one small fragment left behind will give growth to an annoying island of the nail.

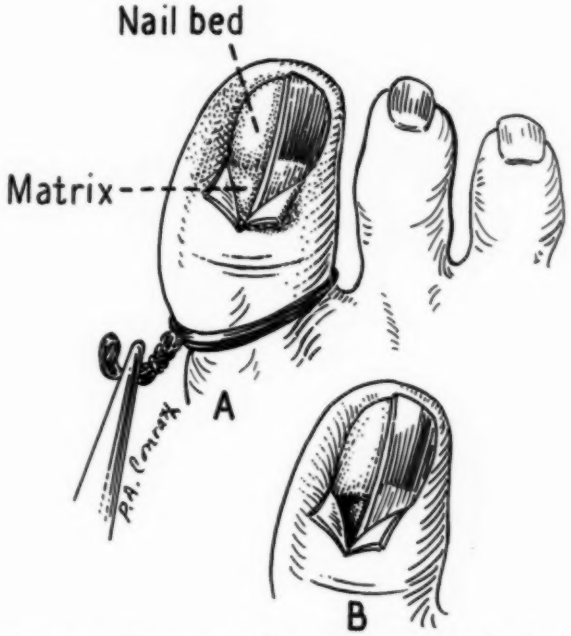


Fig. 10. A. Exposure of the nail-bed and matrix after removing strip of nail. B. The matrix has been curetted away. (Note: the illustrations show the removal of too much nail.)

The matrix is a shiny dense epithelial tissue with a semicartilaginous consistency. It extends proximally about 8 mm. from the interphalangeal joint, distally to the distal margin of the lunula, and laterally well under the lateral exposed flap. (Fig. 10.) The curetting should be quite vigorous as removal of the matrix is difficult, and particular care should be taken in the furthermost corner where the soft tissues give little support against which to work the curette. The matrix lies so close to the periosteum of the distal phalanx, that I never feel certain that it has been completely removed unless I feel the curette grating against the bone. The nail-bed is not disturbed.

5. The wound is thoroughly swabbed with a cotton applicator soaked in 95 per cent phenol, and then with alcohol. This is done to destroy any fragment of matrix that may have become implanted in the wound.

6. It is unnecessary to remove any of the hypertrophied parts or granulations, unless they interfere with proper self-

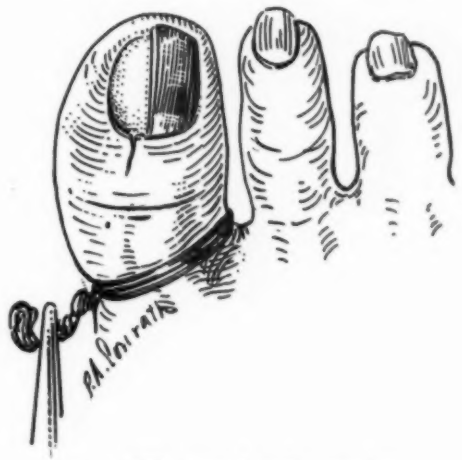


Fig. 11. The completed operation.

closure of the wound. Once the offending nail-edge is removed, these tissues will quickly resume a quite normal appearance. If considerable infection is present, I generally insert into the corner under the flaps a small wick of iodoform gauze. More often, however, no drainage material of any sort is used. No special wound closure is necessary. The flaps will fall back into place themselves (Fig. 11), and any dead space will be eliminated by the application of a tight dressing.

7. The wound is first covered by a layer or two of petrolatum gauze, snugly encircling the terminal phalanx. A few small pieces of plain gauze are then similarly applied, and several turns of a roller bandage made. The tourniquet is now cut, starting considerable fresh bleeding, but this is promptly controlled by completing the bandage. It is important to draw the bandage tightly when encircling the terminal phalanx. There is practically no danger of cutting off the circulation unless the tight turns are made at the base of the toe. The other toes are excluded from the dressing, and it is seldom necessary to include any of the foot. The clean sock is applied, and the old shoe or slipper which the patient brought with him is cut out to fit the configuration of the bandage.

Postoperative Care. Immediately after the operation the patient is instructed to go home and is told not to walk for the rest of that day. So little pain has been encountered even during this day that I have not found it necessary to administer sedatives. Occasionally there is a slight oozing of blood through the dressings, which is of no significance, but if the patient is prepared for it, he will not be alarmed. On the day following operation, he wears his cut-out shoe and is permitted to walk and resume as much of his normal occupation as he desires.

On the second day after operation, the patient returns for his first dressing, at which time the blood encrusted dressings are removed; if a drain has been inserted, it too is removed; a smaller petrolatum gauze dressing is then applied. It is not necessary to dress the toe again until the sixth or seventh postoperative day, when healing is well progressed. At this time, two or three 2 by 0.5 cm. strips of adhesive tape are applied directly to the wound in such a way as to pull the lateral flap in the direction of the cut nailedge. A small dry dressing is applied over this. Thereafter, dressings of adhesive tape are applied every three or four days until complete healing has occurred. Beginning about the eighth day, the patient makes a daily attempt to wear his regular shoe.

TREATMENT OF SPECIAL CASES

Operations on Bilaterally Involved Toe-nails. When one toe has both sides involved, each side of the toe is treated exactly as described as for the one side. This leaves a strip of nail as the center, the width of approximately one-third of the normal nail. In this case, the medial flaps of each side will combine to form one central flap. It is needless to state that when one side of the toe is so advanced as to merit operative procedure, the same procedure should be performed on the other side, even if the lesion be in an early stage and probably amenable to conservative treatment. Five patients had operations on both sides of one toe.

Operations on Both Great Toes. There seems to be no reason why both feet cannot be operated on at the same sitting. This was performed four times, with little extra incapacitation to the patient. In this case, if one toe is involved so that conservative treatment is advisable, operation should be performed only on the toe where the lesion is more advanced.

Ingrown Toe-nails in Diabetic and Arteriosclerotic Patients. In these patients, operative measures are contraindicated. Scrupulous foot care is imperative. Every effort is made to treat the lesion conservatively, even if it means a weekly visit for the remainder of the patient's life. One might anticipate a case of such persistence that the repeated soft part infections would entail a greater risk of gangrene than a brief and careful minor operative procedure. In such a hypothetical case an operation might be considered, but only after very careful circulatory studies have been made. In such a hazardous event, some variations of the operative technique and postoperative care would be necessary.

RESULTS OF OPERATIVE TREATMENT

Twenty-nine ingrown toe-nail operations were performed on twenty patients in accordance with the described technique. All were followed for at least six months, some as long as eighteen months. In each case there was a complete and satisfactory cure.

None of the patients complained of severe pain at any time; whatever pain was present was usually confined to the day of the operation. All were able to walk on the day following operation. Adhesive tape was uniformly applied on the sixth or seventh day. In the average patient the regular shoe could be worn on the tenth day, and complete healing of the wound was present on the sixteenth day. One patient was unable to apply his shoe until the twentieth day and healing took twenty-nine days, but this case was complicated by the pres-

ence of an infection overlooked before operation.

There is nothing remarkable about this particular series of cases, since many of the other methods seem to have produced similar uniformly good results. The real test of the value of the procedure is whether or not it has any of the objections previously mentioned. We shall answer them in order: (1) There is very little disfigurement to the great toe (Fig. 12). (2) The time of healing is quite short, as short as any other operative procedure of a similar nature. (3) The procedure is very simple and can be applied by the average practitioner. (4) Pain is surprisingly little. (5) The patient is ambulatory except for the day of operation. No other operation has this advantage. (6) Cure seems to be permanent in spite of the great variety of footwear worn by these patients postoperatively.

CONCLUSIONS

1. The main underlying causes of ingrown toe-nail are ill-fitting footwear and improper cutting of the nails.

2. The almost exclusive occurrence of ingrown toe-nail on the great toe is probably due to the prominence of that toe on the foot, the relatively wide nail-walls, and the great use of this toe in walking.

3. The symptoms and signs of ingrown toe-nail are conveniently divided into three stages: (a) inflammatory redness and swelling, (b) inflammatory secretion, and (c) granulation tissue formation.

4. An attempt is made to classify and discuss the various methods of treatment.

5. It is possible to simplify treatment by confining it to two types of procedure: a conservative method to handle early cases and cases not amenable to operative procedures, and a radical method for handling the more advanced cases.

6. The use of cotton packing, if properly applied, is one of the simplest and most effective means of conservative treatment.

7. Radical operation is greatly simplified in the method of Winograd, which the writer uses with a few modifications in those cases not recommended for conservative treatment. The detailed technique is described.

Fig. 12. Photograph showing the appearance of the great toe six months after operation. The tibial nail border has been marked with ink.

8. Radical treatment is based on well confirmed physiological principles of nail growth. Since the method described allows the patient to be ambulatory throughout, and since a lasting cure has always been obtained, it is advocated if there is any doubt as to the efficacy of conservative treatment.

SUMMARY

Extensive review of the literature and considerable clinical experience with the handling of ingrown toe-nails have prompted the writer to summarize the present status of the subject, and to outline a simplified mode of management that will be applicable to any stage of the lesion, and that will incur a minimum loss of time to the patient.

The symptoms and signs of ingrown toe-nail are conveniently divided into three stages: (1) inflammatory swelling and redness, (2) inflammatory secretion, (3) granulation tissue formation.

The numerous methods of treatment encountered in the literature can only lead to confusion to the average practitioner of minor surgery. The writer has attempted to facilitate treatment by advocating a simplified mode of management, consisting of a conservative method for handling early cases and cases not amenable to operative procedures, and a radical method for handling the more advanced cases. Both types of treatment allow the patient to be ambulatory.

The use of cotton packing, if properly applied, is one of the simplest and most effective means of conservative treatment, and is recommended for most cases in the first stage and some in the second stage of the lesion.

Radical operation is greatly simplified in the method of Winograd, which the writer uses with a few modifications in all cases in the third stage and most cases in the second stage of the lesion.

With a knowledge of the main etiological factors, a recognition of the three stages of the lesion, and a thorough acquaintance with these two procedures, it is possible to manage intelligently and adequately the vast majority of the cases of ingrown toe-nail with assurance that the results will be satisfactory and that the incapacitation will not be out of proportion to the apparent severity of the lesion.

REFERENCES

1. Anger, T. Sur l'ongle incarné. *Bull. et mém. Soc. de chir. de Par.*, 15: 594, 1889.
2. Barbette. L'ongle incarné. *Jour. des Conn. Medico-chir.*, 7: 197, 1839.
3. Batchelder, J. P. Inverted or imbedded nail. *New York J. Med.*, 1: 9, 1856.
4. Battams, J. S. The treatment of ingrowing toe-nail. *Brit. Med. Jour.*, 1: 833, 1889.
5. Baumgartner. Die chirurgische Entfernung des Nagels. *Berl. klin. Wchnschr.*, 39: 142, 1902.
6. Bennett, C. F. Ingrown toe-nails. *Internat. Jour. Surg.*, 3: 175, 1890.
7. Black, J. R. Inverted toe-nail. *Cincinnati Lancet and Obst.*, 3: 760, 1860.
8. Bloch, O. Remarques sur "l'ongle incarné" du gros orteil. *Rev. d'ortbop.*, 5: 427, 1894.
9. Bopp, L. P. Ingrowing nail. *Boston Med. and Surg. Jour.*, 78: 362, 1868.
10. Bouchaud. Méthode Curative de l'ongle incarné sans operation. *Arch. gén. de méd.*, 30: 428, 564, 1877.
11. Browne, J. W. Treatment of ingrowing toe-nail. *Brit. Med. Jour.*, 2: 857, 1884.
12. Clark, F. LeG. Ingrowing nail. *St. Thomas' Hosp. Rep.*, 13: 1, 1883.
13. Cotting, B. E. A few fragmentary remarks on the radical relief of infleshed toe-nail. *Boston Med. and Surg.*, 116: 324, 1887.
14. Daniljak, J. Zur Behandlung des Haematoma subunguale des Unguis incarnatus und des Panaritium unguale. *Zentralbl. f. Chir.*, 56: 2059, 1929.
15. Dardignac, J. J. A. Réflexions sur l'ongle incarné. *Rev. d. chir.*, 15: 24, 513, 1895.
16. Dolan, H. S. The management of the ingrowing toe-nail. *Can. Med. Assn. J.*, 32: 298, 1935.
17. Dowd, C. N. Report of twenty-nine cases of ingrowing toe-nails operated upon by Anger's method. *Med. Rec.*, 43: 472, 1893.
18. Dugan, W. C. Ingrowing toe-nail and the best method of treatment. *Am. Pract. and News*, 4: 295, 1887.
19. Emmert, C. Zur Operation des eingewachsenen Nagels. *Zentralbl. f. Chir.*, 11: 641, 1884.
20. Fanning, W. A. Ingrowing toe-nail. *Med. Rec.*, 15: 238, 1879.
21. Folts, D. V. Infleshed toe-nail. *Boston Med. and Surg. Jour.*, 88: 280, 1873.
22. Foote, E. M. Ingrowing nail; a comparison of the methods of operation. *Med. News*, 74: 200, 1899.
23. Gallway, J. B. An easy and efficient mode of treating inversion of the nail of the great toe. *Lancet*, 2: 160, 1856.
24. Gerster, A. G. Ingrowing toe-nail. *Internat. Clin.*, 1: 218, 1893.
25. Godman, J. D. Remarks on inverted toe-nail. *Phila. J. Med. and Phys. Sc.*, 12: 339, 1826.
26. Graham, H. F. Ingrowing toe-nail. *Am. J. Surg.*, 6: 411, 1929.
27. Grant. H. H. Operation for ingrowing toe-nail. *Internat. Clin.*, 4: 215, 1897.
28. Gray, H. Anatomy of the Human Body. 20th ed., Phila., Lea & Febiger, 1918, pp. 963, 1066.
29. Gross, S. D. Operation for inverted toe-nail. *Phila. Med. Times*, 2: 410, 1872.
30. Hickman, J. W. Inverted toe-nail. *Med. and Surg. Reporter*, 40: 527, 1879.
31. Hildebrandt. Ueber das Einwachsen des Grosszehennagels. *Deutsche med. Wchnschr.*, 10: 85, 1884.
32. Hilse, A. Zur Operation des Unguis Incarnatus. *Zentralbl. f. Chir.*, 56: 1094, 1029.
33. Howard, W. R. Ingrown toe-nail; its surgical treatment. *New York Med. Jour.*, 57: 579, 1893.
34. Hukill, W. Treatment of inverted toe-nails. *Med. and Surg. Reporter*, 31: 105, 1874.
35. Hunter, C. T. Treatment of ingrown toe-nail. *Phila. Med. Times*, 9: 201, 1879.
36. Ingrowing toe-nail. *Lancet*, 1: 747, 1869.
37. Joseph, S. W. I. The treatment of ingrowing toe-nail. *Brit. Med. Jour.*, 1: 995, 1889.
38. Kelly, S. B. Ingrowing toe-nail. *Boston Med. and Surg. Jour.*, 78: 311, 1868.
39. Kenerson, V. Operations for ingrowing toe-nail. *New York Med. Jour.*, 82: 682, 1905.

40. Keyes, E. L. The surgical treatment of ingrown toe-nails. *J. A. M. A.*, 102: 1458, 1934.
41. King, T. D. A new method for the treatment of ingrowing toe-nails. *Med. and Surg. Reporter*, 51: 733, 1884.
42. Klagas, F. Die Behandlung des eingewachsenen Nagels mittels Silberblechschienchen. *Zentralbl. f. Cbir.*, 60: 1762, 1933.
43. Krecke, A. Ueber den eingewachsenen Nagel. *Münch. med. Wchnschr.*, 80: 1426, 1933.
44. Kurtzahn. Ueber die Behandlung des eingewachsenen Nagels. *Deutsche Zeitschr. f. Chir.*, 218: 411, 1929.
45. Lawson, F. B. A new operation for the radical cure of ingrowing toe-nail. *Med. Rec.*, 5: 511, 1871.
46. Lefebvre, C. Cure radicale de l'ongle incarné. *Gaz. d'hôp.*, 98: 390, 1925.
47. Leonard, J. P. On fungus ulcer of the toe, or that disease usually styled inverted toe-nail. *Boston Med. and Surg. Jour.*, 34: 76, 1846.
48. Loewe, O. Behandlung des eingewachsenen Nagels. *Münch. med. Wchnschr.*, 61: 1178, 1914.
49. Lovegrove, C. Treatment of inversion of the toe-nail. *Lancet*, 2: 250, 1856.
50. McWilliams, C. A. The treatment of ingrowing toe-nail. *New York M. J.*, 77: 1115, 1903.
51. Meigs, C. Cursory remarks on inverted toe-nail. *Phila. Jour. Med. and Phys. Sc.*, 2: 265, 1821.
52. Melion, J. W. Ueber eingewachsene Nägel. *Oesterr. med. Wchnschr.*, p. 1539, 1846.
53. Miall, P. Treatment of ingrowing toe-nail. *Brit. M. J.*, 2: 922, 1886.
54. Mickel. Behandlung des eingewachsenen Nagels. *Deutsche med. Wchnschr.*, 51: 443, 1925.
55. Moher, J. J. E. Treatment of ingrowing toe-nail. *Med. Rec.*, 39: 151, 1891.
56. Morris-Jackson. Human Anatomy. 9th ed., Phila., 1933. Blakiston, p. 75.
57. Moser, E. Operation des eingewachsenen Nagels. *Med. Klin.*, 14: 789, 1918.
58. Néret. Nouveau procédé pour l'arrachement de l'ongle incarné. *Arch. gén. de méd.*, 2: 202, 1838.
59. Ney, G. C. An operation for ingrowing toe-nails. *J. A. M. A.*, 80: 374, 1923.
60. Nicaise, E. De l'ongle incarné. *Rev. de Chir.*, 13: 945, 1893.
61. Nicholson, B. On a cause of ingrowing toe-nail. *Med. Times & Gaz.*, 2: 222, 1866.
62. Nisi, G. Trattamento incruento dell' unghia incarnata. *Il Policlinico*, 37: 1168, 1930.
63. Pancoast, J. Inversion of the toe-nail. *Phila. Med. Times*, 1: 258, 1871.
64. Park, R. Ingrowing toe-nail. *Med. News*, 63: 602, 1893.
65. *Ibid.* Further Suggestions for Relief of Ingrowing Toenail. *Med. News*, 64: 110, 1894.
66. Petersen, F. Zur Operation des eingewachsenen Nagels. *Deutsche med. Wchnschr.*, 10: 196, 1884.
67. Piersol. Human Anatony. Ed. 7, Phila., 1919. Lippincott, p. 1395.
68. Porter, I. G. On incurved toe-nail. *Am. J. M. Sc.*, 39: 124, 1860.
69. Porter, F. J. W. Ingrowing toe-nail. *Brit. M. J.*, 2: 476, 1901.
70. Poynder, G. F. The treatment of ingrowing toe-nail. *Brit. M. J.*, 1: 995, 1889.
71. Purckhauer, H. Zur Behandlung des eingewachsenen Nagels. *Münch. med. Wchnschr.*, 72: 418, 1890.
72. Quénu. Des limites de la matrice de l'ongle; applications au traitement de l'ongle incarné. *Bull. et. Mém. Soc. de Chir. de Par.*, 13: 252, 1887.
73. Reschke, K. Zur Behandlung des Unguis Incarnatus. *Zentralbl. f. Chir.*, 55: 2435, 1928.
74. Richardson, B. W. Treatment of ingrowing toe-nail. *Asclepiad*, 7: 334, 1890.
75. Robbe. De la cure radicale de l'ongle incarné par le procédé de M. Dupuytren. *Arch. Gén. de Méd.*, 11: 432, 1826.
76. deRoulet, A. An operation for ingrown nail. *Am. J. Surg. & Gynec.*, 17: 82, 1903.
77. Schlegtendal. Zur Behandlung des eingewachsenen Grosszehennagels. *Deutsche med. Wchnschr.*, 10: 666, 1884.
78. Scougall, S. H. The treatment of ingrowing toe-nail. *Med. Jour. Australia*, 1: 480, 1934.
79. Secretan, H. Nouveau procédé pour l'extirpation des ongles incarnés. *Rev. med. de la Suisse.*, 6: 759, 1886.
80. Stewart, D. H. The ingrown toe-nail and the coup d'Hache. *New York M. J.* 108: 858, 1918.
81. Stewart P. On inverted toe-nail. *New York J. M.*, 3 s., 1: 367, 1856.
82. Strohe, H. Zur Behandlungs Unguis Incarnatus. *Zentralbl. f. Chir.*, 60: 870, 1933.
83. Taddei, D. La cura radicale dell'unghia incarnata. *La Riforma Med.*, 39: 33, 1923.
84. Tousey, S. Ingrowing toe-nail. *New York M. J.*, 59: 624, 1894.
85. Truehart, C. W. Radical cure of "ingrowing nail." *Med. Rec.*, 5: 196, 1870.
86. Wardrop, J. Treatment of ingrowing toe-nail. *Tr. Med.-Chir. Transactions (London)*, 5: 129, 1814.
87. Webb, G. B. A simple method for the treatment of ingrowing nails. *Med. News*, 75: 829, 1899.
88. Weber, B. Incarnatio unguis; inverted toe-nail. *Cincinnati Lancet & Obst.*, 3: 691, 1860.
89. Winograd, A. M. A modification in the technic of operation for ingrown toe-nail. *J. A. M. A.*, 92: 229, 1929.
90. Winslow, N. A plastic operation for conditions in which complete and permanent removal of the toe nail is indicated. *Bull. School Med. Univ. Maryland*, 18: 12, 1933.

VENOUS THROMBOSIS IN THE LOWER LIMBS: ITS RELATION TO PULMONARY EMBOLISM

JOHN HOMANS, M.D.

Clinical Professor of Surgery, Harvard University; Surgeon to the Peter Bent Brigham Hospital

BOSTON, MASSACHUSETTS

THROMBOSIS in veins—thrombophlebitis is a better name—appears to be on the increase rather than otherwise. It attacks especially patients put to bed on account of injury, operation, childbirth, cardiac disorders and the more debilitating acute febrile diseases. And though Barker has shown that thrombophlebitis can occur in the veins of seemingly normal persons leading an active life, it almost seems as if advances in the care of patients in bed had increased rather than lessened the risk of thrombosis and embolism.

The subject being complicated, attention is invited here to observations reasonably well recognized, particularly those relating to thrombophlebitis in the lower half of the body, the sort which laiety and profession alike are apt to speak of as "phlebitis" and "milk leg" but including, along with the familiar phlegmasia alba dolens, other less obvious and common forms. It is agreed, of course, that somewhat similar processes occur in the heart, the left auricle especially, in the cerebral sinuses, in the portal system, in the vena cava and doubtless elsewhere.

The Process of Thrombosis is not primarily clotting or coagulation but rather the peculiar lesion which begins as a deposition of blood platelets—thrombocytes—upon the wall of a vein. These, having become plastered together and fixed, build out into the current a sponge-like structure in which red and white cells become engulfed and which soon occludes the vessel. The foundation or head of Zahn's classic thrombus is white and tough, the body, which may grow along the vessel both toward and away from the heart for a considerable distance, dark and firm; and the tail becomes more and more clot-like, red and soft until it can hardly be distinguished from normal blood. In so far as this soft part extends away from the heart, it merely blocks more of the peripheral venous tree, but if it extends toward the heart, unfixed in the vein and waving in the current of an entering branch, its end is readily torn free from its base to become an *embolus*. The tendency of thrombosis, in extending toward the heart, is to stop abruptly and heal where it meets a vigorous current, but to grow out into a feeble one like the *propagating thrombus* just described, the sort which waves free, ready to become a source of pulmonary infarction or, if large enough, of a fatal pulmonary embolism.

It is a peculiar quality of thrombosis that it develops most readily in a *slow* current. In the complete absence of a current, that is, in a back-water, it does not form.

THE CAUSES OF THROMBOSIS

These will be discussed only very briefly as "influences." Roughly, they are of three sorts: (1) those affecting the wall of the vein; (2) those altering the nature of the blood, and (3) those which slow the circulation, generally or locally.

Disorders of the Vein's Wall. In respect to thrombosis in large veins, the sort chiefly under consideration here, there is evidence that an initial change in the vein's wall is not *essential* and in many cases is very unlikely to be present. In the superficial thromboses, an abnormality of the vein itself is often fairly obvious, especially in varicose veins and phlebitis migrans. In the deep thromboses, the question of such an abnormality is unsettled, yet in the case of phlegmasia alba

dolens it has seemed to many pathologists as if the inflammatory reaction outside the thrombosed iliac vein was often far more active than the clean state of the contained thrombus justified. The writer has demonstrated, in some pelvic explorations for iliac thrombophlebitis of this sort, a violent, non-suppurative inflammation about both vein and artery, the thrombus itself and the intima of the vein appearing quite reactionless.

Disorders of the vein's wall or of the vascular endothelium are obvious in the case of varicose veins and lead to the formation of rather solid thromboses in especially dilated areas; the inflammatory reaction being such as to fix the thrombus rather solidly and prevent embolism. In the case of "phlebitis migrans," often associated with thromboangiitis obliterans, there is again a local cause for thrombosis, an outspoken inflammatory change in the vein's wall. But in the deeper thromboses of the larger veins the matter is not so clear. A perivenous inflammation in deep iliac thrombophlebitis, that is, phlegmasia alba dolens, was long ago described by Cruvelhier, but is by no means always present and if present, may appear to be the effect just as well as the cause of such a thrombosis. It has already been stated that the writer has demonstrated a very obvious exudate, quite as active about the external and common iliac artery as about the corresponding vein, in several explorations of serious, persistent iliofemoral thrombophlebitis. In harmony with this finding, some deep thromboses are ushered in by such severe pain as to suggest an actual arterial ischemia. Indeed the arterial pulse has been known to disappear before the swelling, due to the venous thrombosis, sets in; only to return as the ordinary signs of an iliofemoral thrombophlebitis become established. This series of events has pointed to an irritating lesion about both the great vein and artery, the latter being excited to spasm while the former is becoming thrombosed. Thus it seems as if some such lesion as a deep perivascular lymphangiitis might be the initial event in such cases, an aspect which the writer has dealt with in previous publications.

Disorders of the Blood take the form chiefly of dehydration, such as may result from vomiting, diarrhea, hemorrhage and sweating at the operating table. Bancroft and his co-workers have found a tendency to coagulation, that is, a high "clotting index" in the blood of many patients, and feel that treatment devoted to this factor aids in preventing thrombosis. There is also another, as yet ill-defined, influence associated with trauma, whether in the nature of a crush or fracture or a deliberate surgical operation.

The influence of trauma may be local, as, for instance, upon the femoral vein of a broken leg or upon the great vessels of the calf when the corresponding foot has been injured and immobilized; or it may be general, as in the case of an iliofemoral thrombosis when a skull fracture has been treated by prolonged dehydrating measures.

It appears also that following abdominal, pelvic and even thoracic operations, entirely outside the anatomical factors to be discussed below, there is a tendency to thrombosis after an interval of ten days or more. It has been suggested that under these conditions the muscles of the legs, which undergo a rather rapid atrophy, contribute some product of atrophy to the bloodstream which leads to thrombosis in such regions as are naturally subject to it.

Disorders of the Venous Return may be due to general circulatory failure, to cardiac weakness plus local venous stasis or to local venous stasis alone. It is perfectly clear that whether the general circulation is, or is not, enfeebled, a patient sitting or reclining in bed subjects the return flow from the legs to a decided slowing. The less the legs are used, that is, the more the muscles are relaxed, the less effectively is blood returned toward the heart. Increase intra-abdominal pressure, as by applying a binder to an abdominal wound and especially by intestinal distension, and the veins find it even more difficult to pass blood rapidly toward the heart.

The anatomic peculiarities of the upper femoral and iliac veins are another unfavorable or, more properly, thrombophilic influence. The eddies and cross-currents due to many entering vessels and the peculiar

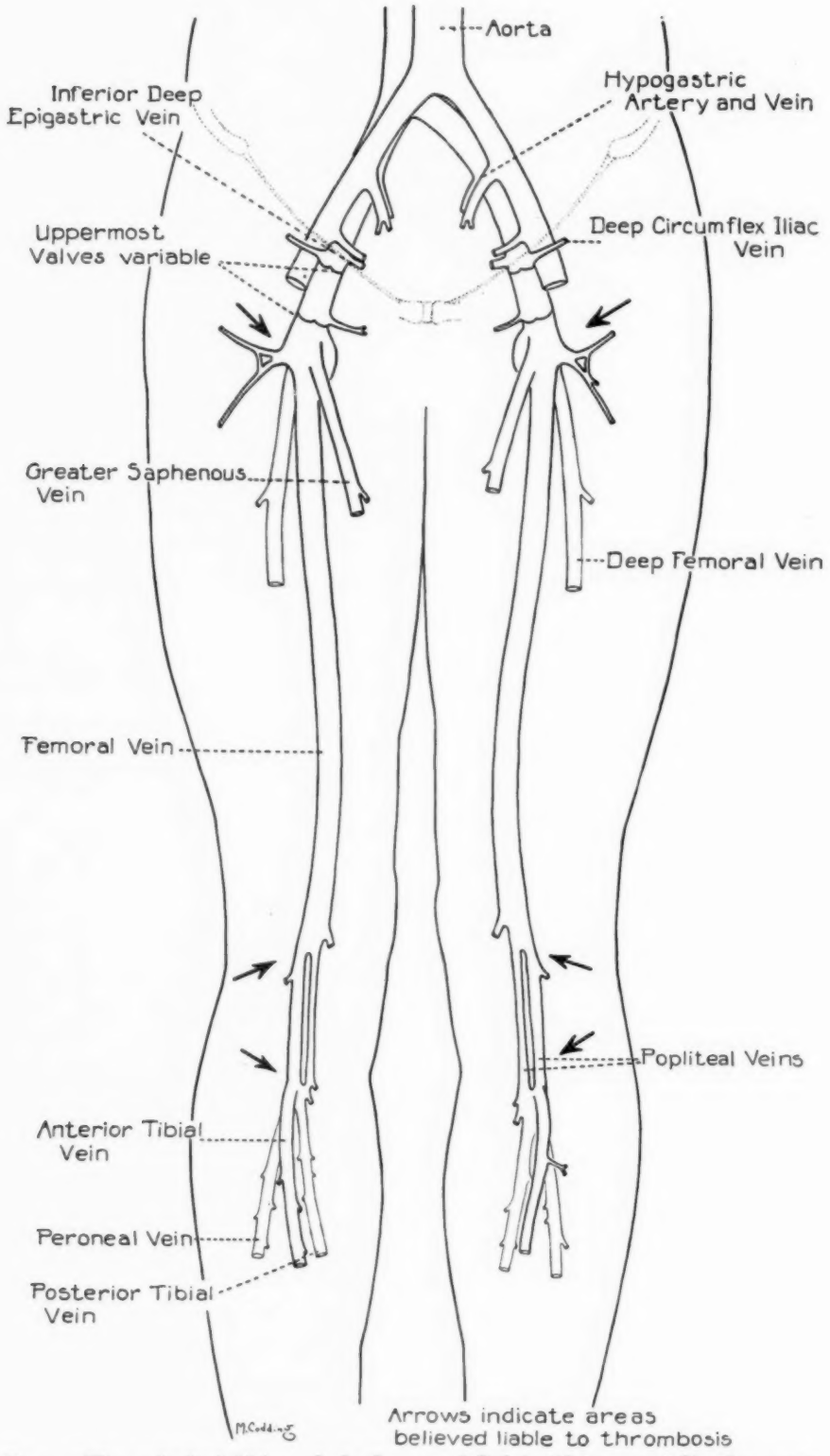


FIG. 1. The principal Veins of the Legs and Pelvis. Note especially the many Branches entering the upper Femoral and External Iliac Veins in the Region of the Inguinal Ligament—also those in the Region of the Knee. Arrows indicate the Sites especially liable to Thrombosis.

relation of the great veins to the arteries alike encourage thrombosis.

Both external iliac veins pass beneath the corresponding hypogastric arteries to become the common iliacs. And both common iliac veins pass behind the right common iliac artery, the left at almost a right angle.

There are often one or two large valves in the region of the inguinal ligament and the following large veins enter the femoral or external iliac over a rather short distance, namely, the deep femoral, the great saphenous, the inferior epigastric and the deep circumflex iliac. According to Virchow, Aschoff and other students of thrombosis, these peculiarities, plus venous stasis, tend to induce thrombosis to this particular region. The upper limb is not subject to nearly so many thrombophilic influences and hence is very rarely the seat of thrombosis. It should be added that there are great plexuses within the muscular masses of the calf and thigh which are not only liable to stasis and thrombosis but are capable of concealing such a process from even the most skillful examination.

To sum up the factors which favor thrombosis, there is: first, inflammation of the vein's wall, a matter of great potential importance, as yet little understood, but as to which there is some positive evidence; second, depletion of the circulation fluids, important but preventable; and, third, slowing of the venous return from the legs, which may be due to an enfeebled circulation, the reclining position in bed and increased abdominal tension, any or all of which influences are aggravated by those local anatomic peculiarities which favor thrombosis in the upper femoral and iliac vein.

VARIETIES OF THROMBOPHLEBITIS

The influences described above are related in various ways to the several varieties of thromobophlebitis distinguishable today, namely, deep (femoro-iliac) thrombophlebitis, or phlegmasia alba dolens, deep peripheral thrombophlebitis or thrombophlebitis among the muscles of the calf, thrombophlebitis in varicose veins and thrombophlebitis in the (non-varicose) superficial veins.

Deep (femoro-iliac) Thrombophlebitis;

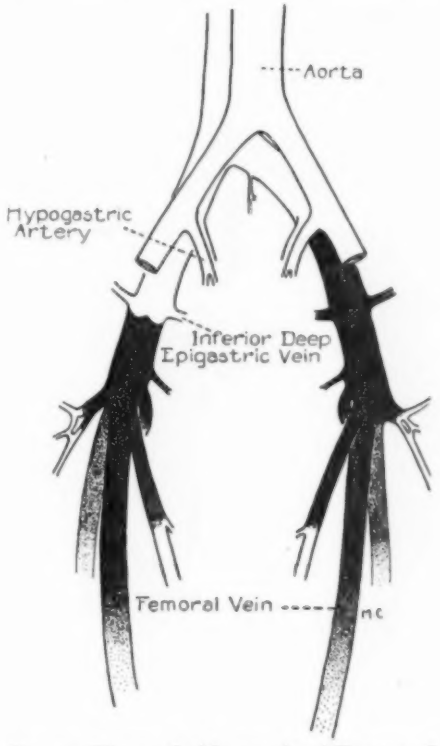


Fig. 2. The probable proximal Extent of a bilateral ilio-femoral Thrombophlebitis. On the left side, the Process reaches the Crossing of the Hypogastric Artery. On the right, it reaches the Inguinal Ligament. Distally, the Extent of the Thrombosis is indicated as being indefinite.

Phlegmasia Alba dolens; Milk-leg. This is the common form with which every one is familiar. Except after child-birth, when it occurs in young women, it is most often seen in middle life and beyond. Thrombosis seems to begin in the upper femoral and external iliac vein. It may extend higher, even into the vena cava, and it occupies the lower femoral tree downward for a variable distance. Probably the severity and duration of the swelling depend upon the amount of venous system which is occluded. That is, the less extensive the thrombosis, the more quickly established and successful is the collateral circulation; and the longer the closed section of vein, and the farther toward the periphery

its tributaries are thrombosed, the more seriously is the return circulation from the whole leg delayed. Swelling, not blueness, seems to measure the severity of this process. There appear to be instances of thrombosis so localized as to cause no swelling of the corresponding leg, yet capable of letting off a fatal embolism. Some of these may arise in active life, but they are most apt to escape attention when they occur while a patient lies in bed, as, for instance in the second leg after the disease has become established in the first. In general, however, a deep femoro-iliac thrombosis causes outspoken swelling.

As regards infection, only very rarely will the thrombus fail to be organized or actually suppurate, throwing off septic emboli.

As regards subsequent function, the softer parts of the thrombus are perhaps absorbed without seriously crippling such valves as are present, and as the most solid portion of a thrombosis is apt to lie in the upper femoral and lower iliac, where few valves are found, it is natural that most patients should recover from a deep thrombosis with little valvular disability that is, with a foot of normal rather than cyanotic color.

A deep thrombophlebitis usually begins with a rise of pulse rate and a little fever. It may be ushered in by pain, even a very severe pain, suggesting, as already explained, arterial stoppage, and referred to the groin, the thigh or even the back of the calf. Or it may creep on in a very silent manner. Swelling starts at the ankle and rather rapidly involves the whole leg and thigh. At first it pits on pressure and may remain soft, though in advanced cases it cannot be indented. Tenderness is most marked at the groin and follows the femoral vessels to the popliteal space or even below. This distribution often falsely suggests involvement of the saphenous vein* which, of course, overlies the femoral for part of its course.

A mild disease lasts ten days to two weeks; a severe one, several months. Subsidence of the fever usually marks the beginning of clinical improvement. Meanwhile the leg is heavy and unwieldy. The skin is pale, occasionally bluish pink and rarely shows a streak or two of lymphangiitis. More often than most persons believe, thrombosis later attacks the second leg, sometimes in so mild a form as hardly to be detectable.

During convalescence, or early attempts to walk, the leg at first becomes a little cyanotic, and the swelling, which may have disappeared, tends to recur. However, it is not edema and congestion which usually trouble people later, but rather the postphlebitic induration and ulceration which the writer has elsewhere described. These troublesome complications, dubbed varicose ulcers by those not familiar with their origin, are often ushered in by the appearance of areas of pigmentation or even local redness and swelling in the lower leg. They may develop at any time, from perhaps three months to twenty years after the initial disease.

Embolism from a femoro-iliac thrombosis is on the whole unusual. Its occurrence, which will be discussed later, can be influenced only slightly by treatment.

Deep Peripheral Thrombophlebitis: Thrombosis in the Calf Muscles. This peculiarly treacherous disease* has received little attention, probably because it is clinically so silent and because pathologists too seldom search the great venous plexuses among the muscles of the lower leg for a cause of fatal pulmonary embolism.

There are present, between and beneath the great flat muscles of the calf, plexuses of veins incompletely used except in times of great activity and capable of nourishing a rather extensive thrombosis without any apparent interference with the venous

* The saphenous system must occasionally be thrombosed, in some degree, as a *complication* of a deep femoro-iliac thrombosis. How often this occurs, the writer has no idea.

* As an entity having a definite symptom-complex, it was described by the writer in 1934. See bibliography.

return of blood. From such a process a propagating thrombus is especially apt to progress into the popliteal vein, whence it waves in the main femoral current ready to break off at any moment. Aschoff appears to believe that most large fatal emboli come from here.

The clinical signs of a deep thrombosis in the muscles of the calf are entirely lacking when the individual lies or even reclines in bed. It is possible that there may be a little discomfort upon forced dorsiflexion of the foot (tightening of the posterior muscles) but it is not yet clear whether or not this is a sign on which to depend. There is no tenderness, blueness, or swelling, unless the patient leaves the legs dependent for some time in the standing (or sitting) position. Then the foot becomes a little blue, the ankle a little swollen. Walking brings out the change even more clearly, together with a moderate local discomfort.

The course of the disease under the usual treatment, or lack of it, is toward apparently complete recovery when the leg is elevated and toward rapid recurrence upon use of the limb. In a typical, rather chronic case, apparent recovery and recurrence may alternate for weeks or months, ending in a fatal embolism. But many thromboses respond favorably to elevation of the leg without immobilization, followed by a gradual resumption of an active life in an elastic bandage.

There is another sort of thrombosis which is thought to be a source of embolism, but as to which little is known. This is the sort which perhaps occurs in the deep pelvic plexuses, about the prostate and bladder in the male and in the uterine veins and pampiniform plexus in the female. Such a process may perhaps remain local or may extend into the common iliac vein. Presumably, insecure thrombi may form in and be detached from this source, but since a diagnosis before embolism is impossible and since the general measures to be directed against thrombosis in the lower limbs applies equally well to the (hypothetical) process in the pelvic veins here described, no further description is required.

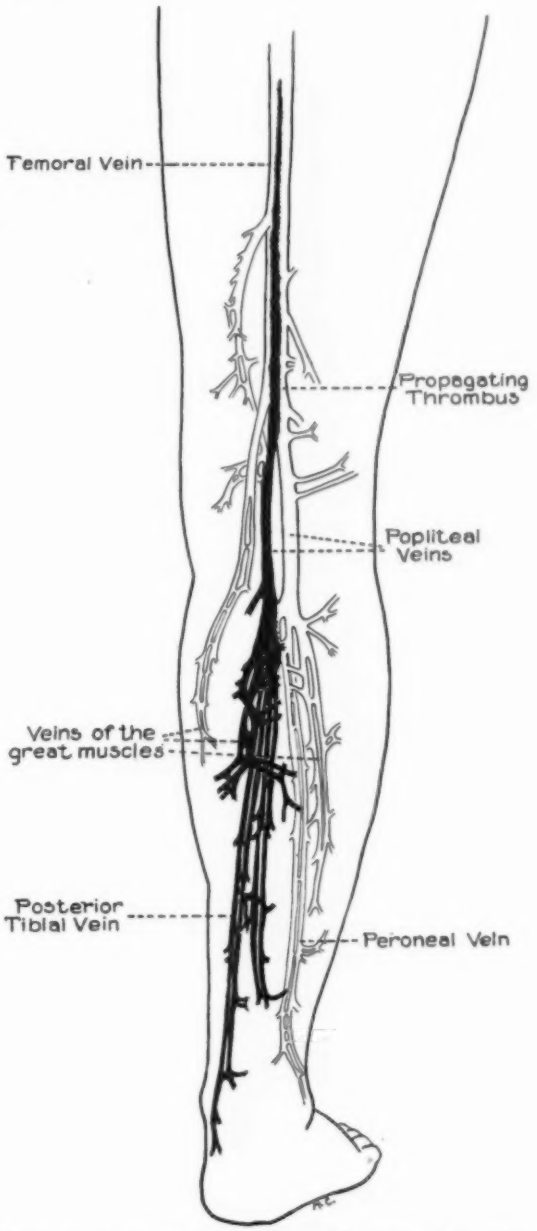


Fig. 3. A propagating Thrombus entering the Femoral from a Group of Veins among the great Muscles of the Calf. Such a Process is difficult to detect clinically. It is almost certain to give rise to a fatal Embolism. (After an autopsy report of a fatal case.)

There remain to be considered the superficial forms of thrombosis, those not primarily under discussion here but of obvious interest by contrast with the deeper processes.

Thrombophlebitis in Varicose Veins, as already explained, is commonly seen when the walls of these vessels have become thin,

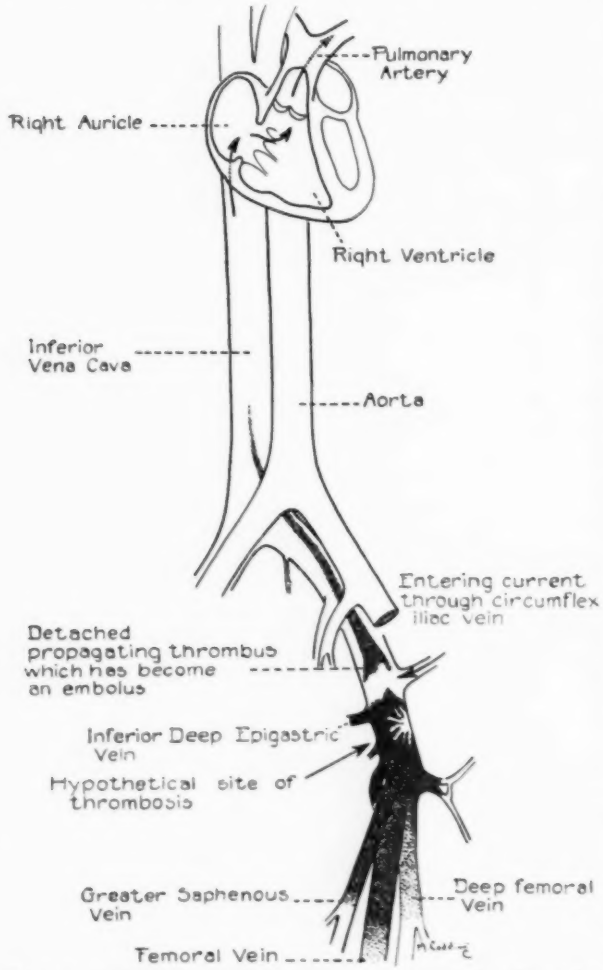


FIG. 4. A hypothetical Representation of the Detachment of an Embolus from an External Iliac Thrombophlebitis. The Course of the Embolus to the Pulmonary Artery is suggested. (The illustrations are from Dr. Robert L. Mason's "Preoperative and Postoperative Treatment," W. B. Saunders Co.)

fibrous and sacculated. It is less apt to occur during life in bed than when the veins are subjected to the pressures forced on them by the erect position. Thrombosis tends to remain confined to the varicose veins and rarely progresses into the femoral or gives rise to embolism. In former times, like other forms of thrombosis, it was treated by long continued rest in bed and the application of ice. It is enough to say of it here that most cases can be treated in an ambulatory way by the application of an elastoplast bandage, only the obviously septic ones requiring rest in bed. To prevent recurrence, high saphenous division plus the retrograde injection of sclerosing chemicals, or even excision, is often necessary. All such measures are exceedingly satisfactory and need not be considered again in the discussion of treatment.

Phlebitis Migrans. This remarkable disease is most often seen as an exhibition of thromboangiitis obliterans. However, it does occur independently, having the earmarks of an inflammatory reaction to a distant focal infection. And although thrombosis necessarily occurs, the primary lesion is probably inflammation of the vein's wall, a true phlebitis. Only a short stretch of vein—a few inches—is at one time involved. Here a tender, pencil-like swelling is present for a few weeks; then it jumps to a higher level, leaving little trace behind. The medium-sized superficial veins of the leg (occasionally the arm or forehead) are affected. Embolism, though rare, does occur.

Traumatic and Infectious Superficial (Non-Varicose) Thrombophlebitis. Although superficial thromboses are rare, they crop up unexpectedly from trivial causes. A tendency to them may perhaps be familiar, and some individuals are subject to an obstinate recurrent sort. An example of a very serious instance is that of a young man (whose father died of pulmonary embolism) who noticed at the end of a long bout of whooping cough a short stretch of thrombosed vein in his right thigh. He applied a piece of adhesive plaster, played a game of squash racquets and suffered a good-sized pulmonary infarction. Afterwards, under treatment by rest and ice-bags, the thrombus spread through most of the large as well as small veins of both legs. Experience with this and some recurrent cases has led the writer to believe that though very active exercise brings a certain risk of embolism, recovery seems to be hastened if, after a few days of rest and *elevation* of the part, moderate exercise is taken in an elastic

adhesive bandage. In other words, given a local thrombosis, dependency of the thrombosed area and local slowing of the circulation encourage an extension of the process.

PULMONARY EMBOLISM

This is a serious problem, especially for the surgeon who operates frequently upon patients beyond middle life, as for instance, upon persons suffering from abdominal diseases and growths. The very dangerous nature of the propagating, insecure thrombus has already been told. Small fragments cause pulmonary infarctions, marked by pleuritic pain and the expectoration of bloody sputum. Large emboli cause sudden death. Between these two extremes are many intermediate states. Infarctions may procede a fatal embolism or may be followed by recovery. In the former case, a propagating thrombus in a large vessel is probably present. In the latter, an embolus is perhaps detached from a smaller vein. It has often seemed as if a whole thrombus was cast off at times, leaving nothing behind, but this may really mean an inadequate search for the source of embolism, especially among the great plexuses of the calf.

Strangely enough, even a large embolus, 6 to 10 inches long, is not always immediately fatal. Such a thrombus has been known to float in the great pulmonary artery without penetrating into either of its branches for hours, days, or even weeks. The patient appears a little blue, especially at first, but may be perfectly comfortable so long as he remains quiet. Finally some change of position or extra exertion causes the embolus to become adherent in one or both of the great pulmonary branches and the patient dies suddenly, so suddenly as to make it seem as if the embolus had just reached the heart. Again, the presence of the embolus in the pulmonary artery will cause agony, breathlessness and cyanosis, much like that of acute coronary disease of the heart's wall, followed by survival for some days.

In recent years, in some perfectly organized clinics, particularly in Sweden, the staff has become expert in making a diagnosis of pulmonary embolism in time to perform Trendelenburg's operation of embolectomy. Some brilliant successes have been scored, but in most hands, failures have been so much the rule as to discourage the belief that lives can often be saved by operative means. Prevention is therefore of prime importance; first, the prevention of thrombosis and, second, the treatment of established thrombosis in such a way as to lessen to the utmost the probability of embolism.

THE PREVENTION OF THROMBOSIS

An earlier discussion should have made it clear that the anatomic and physiologic nature of the venous circulation in the legs and pelvis is such as to encourage thrombosis. Life in bed is another unfavorable influence in setting up what might be called the thrombophilic background, though some thromboses do certainly occur during an active existence. Usually there is added what seems to be the exciting factor, that is, operation, injury, childbirth, debilitating disease, hemorrhage, persistent vomiting, what you will. Such influences are almost always required to induce thrombosis in normal veins, but do not enter into the problem when the vein's wall is essentially abnormal, as in the case of varicosity, thromboangiitis obliterans and local sepsis (otitis media, appendicitis, septic hand.)

Given an injury, major operation or failing heart, the exciting factor is unavoidable. However, its effects can be opposed and modified by treatment. Immobilization favors thrombosis; activity opposes it. A retarded venous return from a dependent part favors thrombosis; an easy return from an elevated part opposes it. Muscular atrophy, an indirect effect of immobility, favors thrombosis; muscular competence opposes it. Increased abdominal tension favors thrombosis; normal

abdominal tension, without compression or distension, opposes it. Prevention requires the sort of effort so lacking in any immediate, obvious beneficial return that few find it possible to keep it up. It should be called "gunning for the hundredth case." For example, don't leave a patient, weakened by operation or disease, sitting up in bed for more than a few hours at a time, unless he or she is suffering so little discomfort as to be able to exercise the legs freely. If help is available, see that some one puts the patient through active exercises for the feet, knees and hips for five to ten minutes at least twice a day. It would be best that patients as to whom you are especially nervous should remain supine and never sit up, except on the way to getting upon their feet. Indeed, unless the patient has a dubious cardiorespiratory system, an elderly man who is subjected to prostatectomy might well be kept head down and feet up for a considerable part of each twenty-four hours during the post-operative period. Why not use the modern bed for all it is worth in preventing thrombosis as well as causing it?

The prevention of increased intra-abdominal tension has been well discussed by Bancroft and his co-workers. They insist that abdominal wounds be so carefully repaired that tight straps and binders are not needed, and regard the prompt restoration of intestinal tone, by the early use of semi-solid or solid food, as an essential point in post-operative treatment. It goes without saying that the surgeon's handling of the abdominal viscera at the operating table may have a far greater influence than any such measures. But it is questionable whether the rather acute and passing phase of abdominal distension is basically as important as the maintenance of a proper fluid balance, a matter which of late years has been very fully dealt with by Coller and his associates. If only one set of measures were available against thrombosis, that should include routine elevation of the legs above the body and active exercise.

TREATMENT OF ESTABLISHED THROMBOSIS

When it comes to treatment, the various sorts of thrombosis require, respectively, slightly different management. The deep thromboses should be placed in one group, the superficial in another.

Treatment of Deep (Femoro-iliac) Thrombophlebitis. Once phlegmasia alba dolens has set in, there is every reason to believe that a solid thrombus occupies the upper femoral and more or less of the iliac vein on one or both sides. The only question is whether at the proximal end of the process there is a propagating thrombus threatening embolism. Usually there is not, but even if there were, sooner or later embolism must occur irrespective of treatment. Moreover, since thrombosis is favored by a slow current, the more briskly venous blood can be passed into the main channel *proximal* to the thrombus, through collateral vessels, the less likely is a propagating, insecure extension of the thrombus to form. Elevation and, if possible, moderate muscular exercise of the affected limb will not only add no new danger, but are probably of real advantage. The importance of draining the limb of fluid has already been urged by De Takats, but the matter of exercise goes even further than this. It should encourage a collateral circulation and check the extension of thrombosis, especially its most dangerous phase. If possible, then, the leg or legs should at all times be elevated above the body, and for at least part of the day, the foot of the bed should be raised above the head. If the leg as a whole can, with assistance, gently be moved, very well. If not, at best the muscles can be tightened by groups without active movements. Massage can help little and may harm the tissues.

When the acute stage of phlegmasia alba dolens is over—fall of temperature to normal, relief of discomfort, softening or disappearance of edema—more active exercise in bed should precede getting up. This, of course, is what most doctors would

consider, and rightly so, I think, the dangerous time, but what advantage can there be in yet longer immobilization? It only puts off, without preventing, the fatal embolism, if such is threatened. So there should be urged, when the leg *appears* normal, the application of a semi-elastic bandage and guarded exercise in the standing position. First, a few minutes of exercise followed by elevation; the periods of exercise being gradually prolonged if the leg does not swell or become cyanotic. The one bad influence, of course, is a dependent position for the leg without exercise. In the end, some collateral veins will perhaps have become dilated, even varicose. In any case, an elastic stocking or bandage may be needed for many months.

The Treatment of Deep Peripheral Thrombophlebitis, that is among the great muscles of the calf, perhaps the most dangerous but least understood of all thromboses, is made especially difficult by the impossibility of measuring the full extent of the process and of deciding whether a propagating thrombus is present. However, if an individual who is suspected of harboring the disease—a little discomfort, blueness and swelling at the ankle on walking, all signs disappearing on rest in bed—suffers a recurrence after one period of rest in bed, it should be realized that a dangerous propagating thrombosis (Figure 3) is probably present, and the femoral vein ought to be divided. Whether or not a long propagating thrombus is present, this step ought to prevent embolism, and in any case appears to cut short the disease. Try, then, ten days of elevation of the leg without bandaging; follow this with gradually increasing exercise in a bandage, and, if no recurrence follows, call the patient cured. Otherwise divide the femoral vein distal to the profunda.

The Treatment of Thrombophlebitis in Normal (Non-Varicose) Superficial Veins. Whether or not the process behaves like a true migrating thrombophlebitis, the patient should be kept for a few days in bed with the foot higher than the head. Without bandaging and especially without an icebag (but applying local heat if you like), you will see the thrombosis very rapidly disappear; whereupon a semi-elastic bandage should be applied and the patient should make an attempt to get about. In some instances, a trial may perhaps be made of a semi-elastic (elastoplast) bandage from the start, but this only in case the patient's reaction to a previous phlebitis is known. The best results are likely to result from five to seven days elevation, followed by bandaging and exercise.

The Treatment of Thrombophlebitis in Varicose Veins has already been touched upon, and is only referred to here to make one point. Though many cases respond well to ambulatory treatment, there are some which clearly require elevation followed by high division (with or without the retrograde injection of sclerosing chemicals), or even excision. In other words, to shorten the disease, but especially to prevent recurrence, radical treatment is often required and is very satisfactory. In those rare cases in which repeated infarction is a feature, it is essential.

SUMMARY AND CONCLUSIONS

The influences, anatomic and related to life in bed, consequent upon many diseases, injuries and operations, which lead to thrombosis in the lower half of the body, are fairly well recognized today. Many of them are unavoidable, but most of them can be robbed of some of their bad effects by prophylactic treatment.

Thrombosis once established should no longer be treated by the familiar ice-bag and immobilization but by real elevation followed early by gradually increasing exercise. Embolism is more likely to be prevented by forestalling the formation of the dangerous propagating thrombus than by apprehensive immobilization. A further study of deep peripheral thrombophlebitis should be made by both physicians and

pathologists. By such means the apparent tendency today to increase in the incidence of thrombophlebitis may be reversed.

REFERENCES

1. Barker, N. W. Primary idiopathic thrombophlebitis. *Arch. Int. Med.*, 58: 147, 1936.
2. Zahn, F. W. Untersuchungen über thrombose. Bildung der thromben. *Virch. Arch.*, 62: 81, 1875.
3. Homans, J. Thrombophlebitis of the lower extremities. *Ann. Surg.*, 87: 641, 1928.
4. Homans, J. The operative treatment of phlegmasia alba dolens. A preliminary report. *N. E. J. Med.*, 204: 1025, 1931.
5. Homans, J. Thrombosis of the deep veins of the lower leg, causing pulmonary embolism. *New Eng. J. Med.*, 211: 993, 1934.
6. Cruveilhier, Jean. Anatomie pathologique du corps humain. Paris, 1829–1842. Ballière. 2: Book xxvii.
7. Virchow, R. L. K. Thrombose und emboli (1846–56), in classiker der medizin. Leipsic, 1910. J. A. Barth, Vols. 7 and 8.
8. Aschoff, L. Lectures on pathology. New York, 1924, Ch. xi.
9. Bancroft, F. W. and Stanley-Brown, Margaret. Postoperative thrombosis, thrombophlebitis and embolism. *Surg., Gynec. & Obst.*, 54: 898, 1932.
10. Bancroft, F. W., Stanley-Brown, M. and Quick, A. J. Postoperative thrombosis and embolism. *Am. J. Surg.*, New Series, 28: 648, 1935.
11. Maddock, W. G. and Coller, F. A. Water balance in surgery. *J. A. M. A.*, 108: 1, 1937.
12. deTakáts, G. The management of acute thrombophlebitic edema. *J. A. M. A.*, 100: 34, 1933.

Myeloma is a rare malignant tumour, nearly always occurring at multiple sites, and favouring the mid-shafts of long bones, especially the humerus and femur, vertebrae, ribs, and flat bones of the skull. It is found chiefly in middle-aged men.

From—"Elements of Orthopaedic Surgery" by N. Ross Smith, F.R.C.S., and R. C. Elmslie, F.R.C.S.

OSTEOMYELITIS IN COMPOUND FRACTURES*

ROBERT H. KENNEDY, M.D., F.A.C.S.

Surgical Director, Beekman Street Hospital; Associate Professor of Clinical Surgery, New York Post-Graduate Medical School, Columbia University

NEW YORK CITY

DURING most of man's existence compound fractures have commonly resulted in the death of the individual. Before the period of Lister in the middle of the last century, because surgeons were unable to cope with infections of all kinds, they resorted frequently to amputations in these patients. This operation of itself carried a high mortality. Up to Lister's time any patient who recovered after a compound fracture, no matter what the deformity and disability, was considered to have done well. How much progress has been made since that time and what is the ideal of treatment at present?

The only large series of results in this country which I have found is from the Surgeon General's Report of the World War.[1] One can say that, due to the exigencies of the time, the equipment, moving from one hospital to another, and the seriousness of the original injuries, these results would not be representative of civil practice. On the other hand, treatment was rendered largely by men who had become particularly interested in the problem, a situation which is often not true in civil surgery. I question whether the results would be appreciably improved if a similar group of fractures in civil life were to be analyzed. According to this report on compound fractures of the femur eight years after injury:

2669 of 5138 patients were less than 50% disabled.
2469 of 5138 patients were more than 50% disabled.

Of the latter 789 were more than 80 per cent disabled. According to these figures practically one-half of the patients who sustained compound fractures of the femur were found eight years later to be still over 50 per cent disabled; 15 per cent of the total were over 80 per cent disabled. This state of affairs, reported to exist within the last ten years, deserves our earnest attention.

In several types of fractures osteomyelitis may be a complication. It frequently develops in infected open wounds in which there is a fracture. This osteomyelitis usually remains localized but may become diffuse. A simple fracture may have a non-communicating wound of the soft parts, a hematoma, or a superficial death of tissue which becomes infected and by direct extension involves the bone. For example a sharp point of the anterior border of the tibia may exert pressure on the overlying skin, followed by necrosis, infection and osteitis. In the course of treating a simple fracture it is not rare for osteomyelitis to be set up as a result of hematogenous infection from some intercurrent disease, such as acute tonsillitis. This complication needs always to be considered when temperature intervenes in the course of a simple fracture. Osteomyelitis all too commonly follows the production of a compound fracture by open operation on a simple fracture. One series of open reductions in good hands shows an incidence of infection of 14 per cent. What must it be in the hands of those who do an occasional case with personnel not especially trained? We should never subject a patient with a simple fracture to the compounding of an open reduction unless we have confidence in the capability of our entire team and the

[1] The Medical Department of the U. S. in the World War ("Surgeon General's Report,") Vol. XI, Surgery, part 1, Washington, 1927, p. 546 and Table 76.

* Read before the Section of Surgery, New York Academy of Medicine, March 6, 1936.

perfection of all our apparatus. Trust in our own technique is not sufficient. Skeletal traction has improved fracture results, but it has entailed some cases of osteomyelitis at the sites of introduction of tongs, pins and wires. In osteomyelitis from other causes, we must keep in mind the supervention of fracture from stress on a bone which has been weakened by disease, a grave complication against which it should be guarded.

There has been much discussion as to whether fractures heal in the presence of infection. White[2] states that "in the majority of cases union takes place, despite the continuance of the infective process. In fact, the persistence of continuous irritation seems to act often as a potent factor in the production of union." Starr[3] says "this infection with sequestration does not prevent union of fractured bone. Even if the sequestrated area involves the whole thickness of the shaft, if the remaining ends of bone are brought into apposition, union usually results." We believe that these statements correctly appraise the situation. Those who have argued that osteomyelitis was a common basis for non-union have possibly considered treatment of the osteomyelitis more than treatment of the fracture. Unless one pays strict attention to complete constant immobilization of the fracture site in the presence of infection he may expect non-union. If he allows surgical dressings to interfere with immobilization he is not justified in placing the blame for non-union on infection, but must accept the responsibility himself for lack of adequate fracture treatment. Unfortunately many compound fractures with infection are treated as if it were impossible to take care of the wound properly and obtain complete immobilization at the same time.

The only completely satisfactory treatment of osteomyelitis in compound fractures is prevention. This treatment should commence at the place where the injured person is found and should not be postponed until he has reached the hospital or the office. In some way traction splints must be made available on our highways and in industry. Patients with fractures of the long bones of the extremities should not be moved until traction has been applied. Too commonly lifting an unsplinted or improperly splinted person or "jackknifing" him into the rear seat of an automobile makes a compound fracture out of what was previously a simple one. A potential osteomyelitis is then present. Application of a fixed traction splint to an extremity in which a compound fracture is already present is of even greater import, whether or not bone is projecting. There seems to be a fixed idea that care must be taken not to allow projecting bone to return within the soft parts until an injured person reaches an operating room. This is false. Gross dirt should be wiped away, a sterile dressing, if available, put on and traction applied. If the bone disappears within the soft parts nothing has been lost as the patient must be operated on anyway. It is only necessary that someone assume the responsibility of seeing that the surgeon is informed that bone has been exposed. If traction is not applied at the site of accident during transportation the bones may be churned about, contamination of both soft parts and bone increased, and more shock with diminution of tissue resistance produced. We must not forget that the origin of fixed traction transportation was in war time and that practically all the fractures so treated were compound. Further, we should recall that the mortality of fractures of the femur during the World War was reduced from 80 per cent to 20 per cent after the introduction of fixed traction transportation. This method is definitely indicated for the prevention of osteomyelitis in compound fractures. Nurses in the emergency room of all hospitals should be trained to apply fixed traction, if it has not been done before, to

[2] White, J. R. Chronic Traumatic Osteomyelitis. New York, Paul B. Hoeber, 1919.

[3] Starr, C. L. Lewis' Practice of Surgery, Hagerstown, W. F. Prior & Co., Vol II, Ch. 2, p. 38.

all possible fractures as soon as they are seen, without waiting for the arrival of an interne or an outside doctor.

The next important point in prevention is early attention. All patients with fractures are emergency cases the same as with acute appendicitis. In compound fractures the problem is one of life and death and permanent disability. The wound does not need to be of great extent for this to be true. The greater the contamination and the longer the primary treatment is delayed, the greater is the likelihood of bone infection. This is naturally particularly true of gunshot wounds with the probability of carrying clothing into the soft parts. There is no excuse for major fractures being left to the less experienced surgeons or the internes in a hospital, nor should treatment be delayed until *x*-rays are reported on or rounds made the following day. The important treatment, which will prevent catastrophes later, is the first treatment. It is often said that this had to be delayed because of the general condition of the patient. This is rarely true if he has been transported in fixed traction and if active shock treatment has been commenced on arrival at the hospital, whether or not at that moment shock seems to be present. Too often we see delayed shock of a severe nature set in when immediate measures have not been taken to forestall it.

The treatment of the fracture and of the wound must proceed together. While the wound is being attended to, traction must be maintained in the original splint, manually, or by some form of apparatus. If traction has to be released to remove the patient's clothing or for any other reason, manual traction should always be maintained until fixed traction is reapplied. The patient should not be allowed to lie on the operating table without the benefit of traction. Not to use it allows further contamination.

A compound fracture should automatically call for immediate thorough debridement. We think of this procedure as something newly devised during the late war. As a matter of fact Petit[4] described debridement in compound fractures in the first half of the eighteenth century. Investigation would possibly show that it was originated long before this. The question often arises of the necessity of debriding the small wound made by penetration of bone from the inside. If the surgeon fails to do this he must be willing to accept the full responsibility for infection developing later, for osteomyelitis, prolonged invalidism and even death. It is too common to see an apparently incised wound ¼ inch to ½ inch long over the middle of the tibia merely dressed. In most cases this would be successful, but the prolonged disability of osteomyelitis, even if only one case in 100, makes such treatment inadvisable. A few days later there may be a sanguinopurulent discharge and bare bone beneath. Often it requires incisions for drainage, removal of sequestrums and prolonged hospital care, if nothing worse.

In some of these cases operated on in the first few hours we are amazed to find the amount of damage which has been done and the extent of dead muscle which should be excised. In these patients, unless there is contamination with street dirt, it is often good judgment to do a primary closure. In the patients compounded from the outside, one should remove all foreign bodies, blood clot, tissue with ground-in dirt and all tissue which seems too badly damaged to live. But this does not require the over enthusiastic debridements seen in war time. It is rarely required to remove bone, and the resection of muscle and bone should not be of such a nature that a useful limb could not possibly be obtained. If that amount of dead and dying tissue is present, the limb should rather be amputated immediately. In civil life, however, we fear that the common tendency is to remove too little rather than too much. In extremity fractures compounded from the outside, the wound should almost invariably be left open after debridement. In the lower

[4] Heister, L. A. General System of Surgery, Ed. 8, London, 1768.

extremity this should always be done. In the upper extremity there is an occasional patient, whom we feel has not come in contact with street dirt or soiled clothing, in whom the wound can be closed completely. To close the wound partially and drain it is dangerous practice. Pockets may be shut off inside and infection may be introduced from the outside. If after a debridement infection manifests itself free drainage is easily established, inflammatory exudates do not extend because of tension, periosteal stripping is not increased and whatever osteomyelitis occurs usually remains well localized.

Reduction of the fracture is attended to at the same time as debridement and it is determined what type of internal or external fixation is to be used. Whether this is some internal appliance to the bone, or skeletal traction or plaster casing it must be a permanent arrangement which will maintain the reduction. If the after care will require change of dressings the immobilizing apparatus must be so devised that there can be no disturbance of the bony relations during the dressing. If traction is to be used the amount of weight necessary to maintain reduction should be determined at the operating table. These are not cases suitable for gradual reduction over twelve to forty-eight hours. We are dealing with a potentially infected wound and the soft parts and bone fragments must be immediately put to rest and kept at rest. Continuous slight change in position will increase the likelihood of infection becoming established. For the same reason it has to be understood that a later attempt at any closed manipulation of the fragments carries with it grave danger. Adhesive plaster traction or Russell traction should not be used, because of the possibility of slipping of the adhesive plaster with resultant mobilization of infection from change of position of fragments. Complete constant immobilization is the absolute requirement in these patients and this refers to soft parts as well as bone. Of course no massage should be used until there is certainty that asepsis has been established. All of this routine constitutes the preventive treatment of osteomyelitis. I repeat that the only satisfactory treatment of osteomyelitis in compound fractures is prevention.

In spite of all of our efforts suppose that infection occurs. This requires early adequate drainage without disturbing immobilization. Sufficient ventilation of the soft parts will keep the extension of bone infection to a minimum. The bone should not be disturbed, unless fragments are so loose that they are evidently acting as foreign bodies and can be picked out.

Notwithstanding our best efforts osteomyelitis will sometimes become established. Foster[5] reported that following debriding of compound fractures a discharging sinus was present four months after injury in 11.6 per cent of 129 plated cases and in 15.7 per cent of 141 cases treated by traction and splinting. These patients were treated under conditions which are at least as good as anywhere in the United States. In addition some patients will be sent to us with definite chronic osteomyelitis following fracture. These require the greatest judgment and patience in treatment. If the fracture is not solid, effective immobilization must be maintained regardless of other considerations. An *x*-ray picture is of great assistance if there is evidence of sequestrum formation. No attempt should be made to remove a sequestrum until we are quite certain that it is free on all sides. Neither should it be allowed to remain when it is apparently free, on a chance that it will be extruded by itself. It should be removed as soon after sequestration is complete as we believe that the healed bone and involucrum are strong enough to support the part without refracture. If union is not progressing, it may be removed immediately. The surrounding tissue should be disturbed as little as possible and the wound left open

[5] Foster, G. V. *Surg., Gynec. and Obst.*, 56: 529–538, 1933.

Later it may become necessary to obliterate sinuses and cavities. This requires careful excision of the involved soft parts and bone with gentle removal of overhanging bone edges converting cavities into easy slopes so that the soft parts may fall into them. Again leave the wound open and immobilize the part.

In other patients it becomes necessary to remove chronically infected living bone, often to a considerable extent. This requires a sloping removal of the bony walls. Whatever form of after treatment is to be followed, these wounds should usually be left open and soft parts allowed to fall in.

Sometimes with an extensive osteomyelitis, with or without non-union, an amputation may be more advisable than prolonged hospitalization with possibly a limb of slight usefulness when healing has been obtained. This should never be done without the acquiescence of one's confrères.

CONCLUSIONS

In conclusion, I ask for the injured person who has a simple fracture the chance to be transported without a compound fracture being produced; in both simple and compound fractures to have traction splints made available so that further injury and contamination will not occur. I ask for immediate emergency care of compound fractures with debridement by attending surgeons. I ask for continuous effective immobilization of these fractures from the time of injury until union is solid, in spite of the dressings required. I ask for early adequate drainage should infection develop and conservative but thorough treatment should osteomyelitis become established. Time is better spent for the surgeon and patient by attention to the details of early treatment of a compound fracture to prevent osteomyelitis than by the most meticulous care of osteomyelitis after it has developed.

A RECONSTRUCTION OPERATION FOR COMMINUTED FRACTURE OF THE UPPER THIRD OF THE ULNA*

LESLIE V. RUSH, M.D., F.A.C.S. AND H. LOWRY RUSH, M.D., F.A.C.S.

MERIDIAN, MISSISSIPPI

COMMINUTED fractures of the upper third of the ulna usually occur as the result of direct violence, a blow striking the posterior surface of the upper third of the forearm while the elbow is in the position of flexion, the force being directed upward toward the shoulder. The resulting bone injury is an anterior dislocation of the radial head with marked comminution of the upper third of the ulna and wide separation of the fragments. The action of the triceps tendon pulls the olecranon process upward, the shaft and the comminuted fragments being driven anteriorly. Fixation of these fragments by an intramedullary peg is usually unsatisfactory because of the short olecranon fragment and extreme comminution of the shaft.

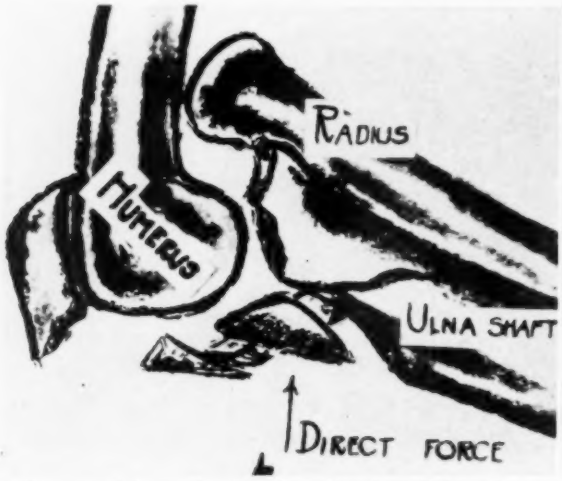


FIG. 1. Schematic drawing showing dislocation, radial head and comminution of ulna shaft.

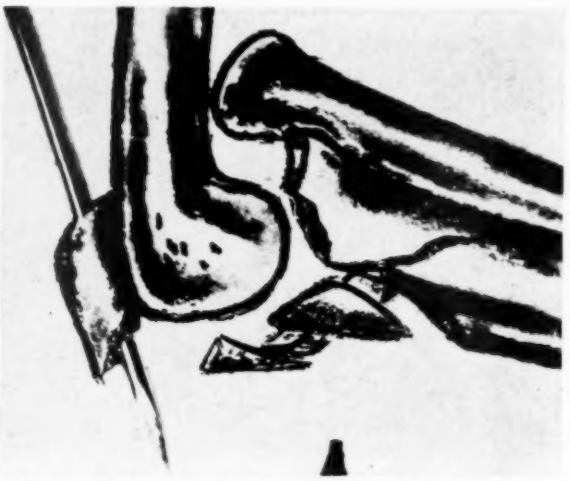


FIG. 2. Schematic drawing showing Steinman Pin directed through olecranon.

It is difficult to maintain adequate fixation by means of wire because of the tendency of the radial head to dislocate anteriorly, carrying with it the shaft and the comminuted fragments. Overcoming this tendency by acute flexion often causes embarrassment of the circulation, as there is usually severe soft part damage. The wire may break or untwist from the extreme tension.

Open reduction with maintenance of alignment and fixation by means of a Steinman Pin has proved most satisfactory, permits early active motion, and facilitates greatly the removal of the internal fixation.

TECHNIQUE

A longitudinal incision, usually about 3 or 4 inches in length, is made on the posterior surface of the forearm, beginning at a point over the triceps tendon and passing downward to expose the fracture site. Firmly attached bone fragments are not disturbed. Loose fragments are removed temporarily and wrapped in a sterile sponge moistened with normal salt solution. The Steinman Pin is then introduced through the olecranon, beginning at the tip and directed in such a fashion that the point will protrude through the mid portion of the fracture plane. (Fig. 2.) The pin is then passed distally in the forearm until

* From Rush's Infirmary Clinic, Meridian, Miss.

the point is introduced into the medullary cavity of the shaft. (Fig. 3.) By bringing the ulna into proper alignment, the radius usually falls into its normal position and is maintained there without difficulty. The comminuted fragments are replaced about the Steinman Pin, filling the gap between the proximal and distal fragments. The comminuted fragments are maintained in position by tying with chromic cat gut, kangaroo tendon, or small stainless steel wires. (Fig. 4.) Periosteum and fascia are approximated with No. 0 chromic cat gut and the skin sutured with interrupted black silk. The Steinman Pin is allowed to protrude for a distance of about three-fourths of an inch, posteriorly. The elbow is dressed at a right angle using an anterior plaster splint.

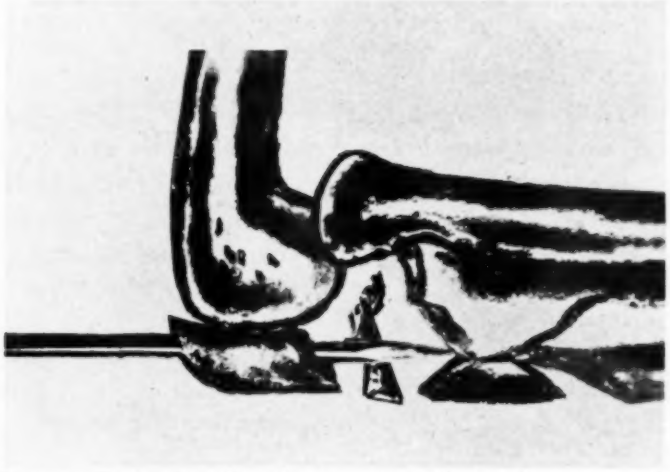


Fig. 3. Reduction has been accomplished—olecranon and Steinman Pin brought into alignment with ulna shaft, point of pin being directed into medullary canal.

After Treatment. Sutures are removed on the ninth day. At that time the protruding end of the Steinman Pin is coated with adhesive plaster to prevent its slipping inside the arm. Active motion is encouraged after two weeks unless there has been severe soft part damage.

Myositis Ossificans is a common complication in this type of injury. Frequent X-ray films are essential. At the first sign of myositis ossificans the arm should be immobilized in acute flexion, which is the most useful position should ankylosis occur. Secondary surgery may be done later, if necessary, to relieve any disability resulting from the myositis ossificans.

Since firm union in this type injury might be delayed, the Steinman Pin is not removed until the *x*-ray film shows evidence of firm union. This usually occurs sufficiently between the fourth and eighth week. Too early removal of the pin will result in an anterior bending of the ulna with forward dislocation of the radial head, and too vigorous active motion of the elbow in hyperflexion will result in posterior bending of the ulna. With the Steinman Pin in situ, active motion can be carried out more vigorously and with less fear of disturbance of alignment.

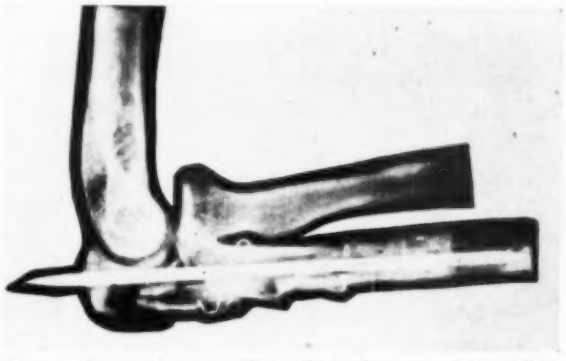


Fig. 4. Lateral *x*-ray film. Complete reconstruction. Position olecranon and shaft maintained by pin. Bone gap filled in by bone fragments fixed around pin by circular stainless steel wires. Pin protrudes for three fourths inch beyond skin.

TREATMENT OF DISTENTION BY CONTINUOUS DUODENAL SUCTION*

MILTON S. WEINBERG, M.D.

NEW YORK CITY

DISTENTION following a surgical procedure has always been a distressing problem to the surgeon. The distention may vary from the so called "gas pains" following a simple appendectomy which the lay friends of the patient tell him to expect, to an acute gastric dilatation, paralytic ileus, or the distention found in peritonitis or bowel obstruction. The degree of distention is independent of the surgical procedure as the most marked distention may follow an operation where the least amount of handling of the viscera has been suffered or even where the viscera hasn't been touched.

The number of explanations for the cause of this condition compares favorably with the number of operations for the radical cure of hernia. Some advance the theory that excessive handling of the viscera is the cause. Toxins have been mentioned as the source of the original disturbance. The anesthetic and the amount of anesthesia has been suggested as a factor in distention. The exposure of the peritoneum to the air is believed by some to be the cause. Since distention is present in various forms of toxemia as well as in cases where no toxemia is present or where no surgical manipulation has been experienced the cause of distention is still obscure.

At the first appearance of distention the normal tonus of the digestive musculature is lost. This tonus is an intrinsic property of the smooth muscle contained in the walls of the gastrointestinal system.

In the normal stomach, the vagi through the plexus of Auerbach stimulate contraction waves similar to peristaltic waves while the splanchnic nerves supply inhibitory impulses. These are essential contributory factors in maintaining normal tonus. Good intra-abdominal tension is a requisite of normal tonus as in an empty stomach the walls are in close apposition.

Since gastric motility is perhaps the outstanding function of the organ, acute impairment of the motor mechanism is a serious handicap. When for any reason the normal tonus is impaired there is first a state of mild atony. By the same mechanism as the paralysis is produced, an unusually large amount of thin watery secretion seems to well forth from the walls of the stomach or intestine which further distends the musculature and accentuates the atony, forming a viscious cycle and peristalsis ceases entirely. As a result we have the acute gastric dilatation or paralytic ileus.

Since this paper is not a treatise on paralytic ileus or acute gastric dilatation it is suffice to mention the symptoms common to both and also common to peritonitis. The most prominent symptom is that of marked distention. The distention may be such as to interfere with breathing and cardiac action, and may be sufficient to cause the wound to rupture and the viscera balloon out through the opening. Peristent vomiting is a constant symptom. At first the vomitus is gastric contents, followed by bile stained fluid, and then by a black or brownish fluid which is present in an unusually large amount. The abdomen is silent with no peristalsis. Various systemic changes have been observed such as elevated temperature, rapid feeble pulse, etc. Often these symptoms occur in a mild degree and only air is belched as the accumulation of the fluid is pushing out the air which may be present there.

In speaking of acute gastric dilatation, Babcock in his latest book gives a prognosis

* The work described in this article was done at the Hahnemann Hospital, Scranton, Pa.

of "50 to 75 per cent, the pulse increases rapidly, and the patient goes into collapse and dies." Thomas R. Brown in Cecil's book on medicine tells of a mortality of 74 of 102 patients. The gravity of the case of diffuse peritonitis is known to all and when this occurs in the aged the prognosis is even worse.

Since the purpose of this paper is to describe the treatment we have used most successfully in the series of cases to be reported it is fitting to discuss the usual treatment of dilatation used previously. Doses of strychnia sulphate 1/30 to 1/20 grain every three hours have been recommended but since this acts through the vagi which are paralyzed by the distention, its effects are useless. Pituitrin and pitressin have been administered with the aim of inducing peristalsis. We often see milk, molasses and turpentine enemas in an attempt to expell gas following a simple appendectomy. The use of the Murphy drip is almost a routine treatment in peritonitis. Decompression of the abdomen is the most important treatment in this case.

We have used a modification of Wangensteen's apparatus for continuous duodenal suction (Fig. 1) with most gratifying results. The relief of the distention by the use of this continuous suction allows peristalsis to resume of its own accord as soon as the reparative processes of the body have allowed the injured member to recover. In the presence of dilatation there is a negative pyloric balance in which more fluid regurgitates through the pyloric ring than goes through in the normal manner. When positive pyloric balance is established fluids will pass through the pyloric ring and pass on through the intestine. This is dependent on normal peristaltic action. When the regurgitant fluid withdrawn exceeds the oral intake we know that there is negative pyloric balance and we allow the tube to remain in place until the regurgitant fluid is considerably less than the oral intake which shows us that peristalsis has been established. Since the continuous suction is present it is possible to give the patients liquids as soon as they have reacted.

The apparatus we are using makes use of

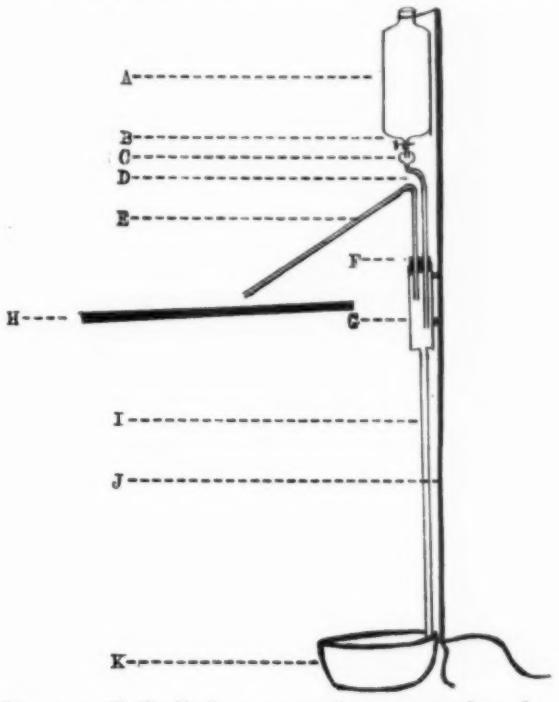


Fig. 1. A, Kelly flask; B, screw clamp to regulate the number of drops; C, Murphy drip bulb; D, ordinary glass drinking tubes; E, duodenal tube from patient; F, two holed rubber stopper; G, Luer syringe barrel where suction is created; H, level of bed; I, rubber tube; J, stand; K, receptacle for gastric and duodenal contents and water.

the principle of indirect suction created in a Luer syringe barrel by water dropping by an open tube. Tap water suspended in a Kelly flask is allowed to drip through the Murphy drip bulb at the rate of 30 to 40 drops per minute. This is carried down to the Luer syringe barrel which is closed by a two hole rubber stopper. One opening is for the tube conducting the tap water from the Kelly flask while the other opening is connected to the Rehfuss tube placed in the patient. The tap water drips through the opening in the syringe barrel and creates the suction which draws the gastric and intestinal contents through the Rehfuss tube into the syringe barrel and out through the opening into a container. That this is suction and not siphonage is proved by the fact that it will raise a column of water 10 inches high. This

suction is not of sufficient intensity to dehydrate the patient.

CASE REPORTS

Case 1. L. G., white female, aged 26, para 11 was admitted to the hospital with a diagnosis of pregnancy for which a cesarean section was required, the conservative Sanger technique being used. The most extreme precautions were taken as far as cleansing the gastrointestinal tract preoperatively. The anesthetic used was nitrous oxide induction and ether. Postoperatively pituitrin, caffeine sodium benzoate and hypodermoclysis were used. Five hours after the operation the patient vomited a small amount of brown liquid. She also complained of interference with respiration, nausea and eructations. Twelve hours after operation the patient was allowed sips of water at approximately four hour intervals. Distention was noticed on the first post-operative day. The patient was allowed to have small quantities of water or tea but vomited twice within a six hour period. Tap water was prescribed by bowel. The distension became progressively worse but the abdomen was still soft. Retention enemas of tap water and the rectal tube were administered but the distention continued and the emesis of green liquid recurred in increasing amounts. Sodium bicarbonate solution in large doses was prescribed in an attempt to change the hydrogen ion concentration of the system but this did not relieve the distension or check the vomiting. A milk, molasses and turpentine enema brought forth good results and the abdomen became much softer. At the same time surgical pituitrin 1 c.c. was given and a rectal tube inserted. This condidition continued with subsidence of the distention following an enema and its reoccurrence with vomiting of increasing amounts of green watery fluid in spite of apparent bowel movements. On the seventh postoperative day, the wound which was apparently healthy opened and the intestines ballooned out. The wound was resutured but the distension was still present and so marked that the abdomen was hard. Milk and molasses enemas along with pituitrin were repeated and although stools were passed the distension and vomiting persisted or in fact increased in severity. Consultation was called and pitressin was substituted for pituitrin with no apparent relief.

On the ninth postoperative day and the second day after the resuturing, the patient presented a very grave prognosis. The temperature was between 102 and 103°F., the pulse rate was between 140 and 150 and the distention and hardness of the abdomen was not affected by any further medication. The vomiting was profuse and protracted.

Since the Wangensteen duodenal suction had never been used before at our hospital, it was decided to make use of it as a last resort. Upon introducing the tube 600 c.c. of greenish bile stained fluid were removed by direct suction. Fluids were given ad lib which amounted to about 500 c.c. in two hours and this in conjunction with the fact that the patient was not stable emotionally, resulted in the patient vomiting the duodenal tube.

The tube was replaced and it was explained to the patient that she wouldn't get well without it. Morphia was given to control the patient and the fluids were limited to 3 ounces per hour. In a period of five hours there was a regurgitation of 510 c.c. The abdomen became soft and the distention disappeared. Ten hours after the tube had been inserted the report for a three hour period was 450 c.c. returned through tube while 510 c.c. were given by mouth indicating that at least 60 c.c. had passed through the pyloric ring and passed through the intestine. To prove that peristalsis had been established we have the passage of large amounts of flatus per rectum and a bowel movement. The improvement proceeded rapidly; so, that after thirty-four hours of treatment, only 370 c.c. had been withdrawn from the stomach in four hours while 900 c.c. fluid was given by mouth thus showing that 530 c.c. of fluid were passed through into the intestine and thus the tube was removed. The temperature at the time of the removal of the tube was 100°F. and the pulse rate 100.

Three days later while the patient was on a soft diet, the temperature rose to 103°F., the pulse rose to 155, and the abdomen became distended and hard. The use of the rectal tube, pitressin, and a milk, molasses and turpentine enema again brought no relief from the distension. The continuous duodenal suction was again applied for thirty-six hours relieving the distention and initiating peristalsis in twelve hours, evidenced by the patient expelling large quantities of flatus per rectum. She was placed on a soft diet twenty-four hours after the tube was removed and went on through

an uneventful convalescence to be discharged twelve days after the suction was first applied.

Case II. G. M. K., white male, aged 58, was admitted with the diagnosis of acute appendicitis. The leucocyte count was 20,600 of which 88 per cent were polymorphonuclear cells. At operation the diagnosis of peritonitis was made, the appendix was removed and a drain inserted. Hypodermoclysis of 500 c.c. of 5 per cent glucose in saline was started while the patient was on the operating table. Pitressin 1 c.c. was ordered soon after the patient's return from the operating room. This was repeated within three hours and continued at intervals of three hours. Retention enemas of 10 ounces of 5 per cent glucose in normal saline were given every five hours. Fluids consisting of small amounts of water were given six hours after operation and were continued. Ten hours after operation the patient expelled gas by mouth and by rectum. Four hours later the patient vomited 4 ounces of bile stained fluid while the abdomen was hard and distended. Pitressin was continued every four hours and the rectal tube was inserted with only a moderate amount of gas expelled. The patient received 42 ounces of fluid by mouth on the first postoperative day at the end of which he vomited 6 ounces of green bile stained fluid. The abdomen was still rigid and distended on the following day when a high compound enema was given with poor results.

At this time the duodenal tube was passed and 200 c.c. of bile stained fluid were removed. On the application of continuous duodenal suction no fluids were obtained but large amounts of gas bubbled through. At the end of two and one-half hours the abdomen was considerably softer and the distension had been markedly reduced. Water passed through the tube and could not be removed by direct suction, showing us that peristalsis had been established. Two ounces of castor oil were introduced through the tube and the tube removed. A high compound enema brought fair results and a large amount of flatus was expelled by rectum. The abdomen remained soft and only moderately distended at times. A pulmonary complication set in on the fourth operative day and the patient died on the fifth day.

In this case it can be seen that the effects of the enemas and pitressin were negligible before the distention was relieved. However, as soon as the distention was relieved the medication took effect.

Case III. M. McD., female, white, aged 7, was admitted with the diagnosis of ruptured gangrenous appendicitis with peritonitis. At operation free pus was found in the peritoneal cavity which necessitated a suprapubic drain. Shortly after operation continuous duodenal suction was applied in conjunction with Murphy drip and Fowler's position. In the first four hours there was a regurgitation of 100 c.c. In the next forty-eight hours just as much fluid was removed by the tube as was given by mouth which was 2385 c.c. the first day and 1860 c.c. the second day. On the third day the patient took 2430 c.c. of fluid by mouth of which only 1375 c.c. were returned through the tube and 1055 c.c. were passed on through the intestines. The apparatus was removed the following morning and the patient proceeded to an uneventful recovery to be discharged on the fifteenth day. At no time did the patient complain of gas pains or vomiting after the tube was placed in position.

Case IV. L. O., male, white, aged 62 years, was admitted with the diagnosis of acute gangrenous appendicitis which on the operating table proved to be the same with a general peritonitis. The duodenal tube was inserted as soon as the swallowing reflex returned and fluids were given by mouth almost immediately. Glucose 5 per cent was given by hypodermoclysis, a Murphy drip of glucose solution was started and the patient placed in the Fowler's position. The Murphy drip was discontinued on the first day because of irritation. In the first six hours the patient took 120 c.c. of water by mouth and the amount withdrawn through the tube was 340 c.c. thus a regurgitation of 220 c.c. In the next five hours 340 c.c. were given by mouth and only 300 c.c. were returned through the tube, and at the same time flatus was expelled by rectum. The duodenal return diminished progressively until the second day postoperatively, when the patient took 870 c.c. of water in nine hours with only 300 c.c. returning, showing that 570 c.c. passed through the intestine. On the evening of the same day the patient passed a small liquid stool with flatus. The tube was removed on the fifth postoperative day.

The patient was continued on a liquid diet until the seventh day when he was placed on a

soft diet. The patient progressed so rapidly that preparations were made for his discharge on the eleventh day. On that day a slight fever was observed which necessitated the patient remaining in bed for another week before he was discharged. At no time was catharsis or enemas required to stimulate bowel movements. Hypodermoclysis was used only once.

Case v. D. M., white, male, aged 16 years, was admitted with the diagnosis of subacute appendicitis. On operation a ruptured gangrenous appendix was found, an appendectomy was performed and a drain inserted. With the exception of gas pains and some emesis the recovery was uneventful and the patient was discharged in thirteen days. Three days after his discharge the patient was readmitted suffering with extreme abdominal pain, tenderness, rigidity and obstipation. The return from the high compound enema was clear and no flatus could be expelled, there was emesis of greenish liquid in large amounts. At operation a pelvic abscess containing three pints of colon pus was opened and drained by suction. The patient was in such poor condition that the wound was closed hurriedly and the case was turned over to the internes to treat to the best of their knowledge. The patient was placed in the exaggerated Fowler's position and intravenous glucose 5 per cent was given as soon as he was returned to bed.

Wangensteen continuous duodenal suction was applied at the same time and tap water was ordered by mouth. In the first three hours the intake by mouth was 350 c.c. while the amount removed through the duodenal tube was 1300 c.c. indicating a regurgitation of 950 c.c. During the next ten hours the oral intake was 660 c.c. while the amount drained from the duodenum was 800 c.c. thus having a regurgitation of 140 c.c. During this time the patient had a bowel movement with no stimulus other than the natural one, and then another one three hours later. The fluids were varied, with ginger ale, orange juice, tea and bouillion being used in place of tap water. Gas was passed freely through both the duodenal tube and through the rectum. At the end of three days the amount of fluids by mouth was 600 c.c. in four hours and the return was only 40 c.c. showing the establishment of peristalsis, so that the tube was removed.

The patient experienced daily bowel movements without the aid of catharsis or enemas. At no time during the convalescence was this patient distended nor did he vomit during the period. He was placed on a soft diet on the fifth postoperative day and was discharged as soon as the wound healed by granulation three weeks after the operation.

Case vi. H. W., male, white, aged 54 years, was admitted with the diagnosis of chronic appendicitis. At operation the diagnosis of carcinoma of the cecum was made and as the malignancy was beyond control the abdomen was closed. In spite of the most extreme preoperative care, which included thorough colonic lavage and pitressin, and postoperative care the patient developed a severe distension in twenty-four hours. Pitressin was given every four hours with very little improvement. The patient was belching gas but passed nothing per rectum. High compound enemas, rectal tube, hot packs to the abdomen and rectified spirits of turpentine by mouth were tried with no apparent improvement in the condition. As the distension increased, there was emesis of large amounts of greenish watery substance.

On the third day when the distension showed no signs of regressing, the duodenal tube was passed and the continuous duodenal suction applied. On direct suction only 60 c.c. of regurgitant fluid was brought up. In the first seven hours there was a regurgitation of 430 c.c. while the patient was allowed 340 c.c. of fluid by mouth which meant that 90 c.c. was succorrhea. In the next twelve hours the patient was given 810 c.c. by mouth of which only 650 c.c. returned indicating that 160 c.c. had passed through the intestine. In the subsequent three hours 450 c.c. of fluid including broth, water and orange juice, were given of which only 25 c.c. came back through the duodenal tube showing a presence of active peristalsis. As this fact was confirmed by the passage of feces and flatus the duodenal tube was removed having remained in situ but twenty-one hours. The pitressin was discontinued on the following day. There was no more distention during the remainder of his stay in the hospital, which was thirteen days.

We feel that in this case the use of the suction and pitressin was admirably combined. Pitressin was given before the duodenal suction was used with no appreciable results, but it seemed to get in its work as soon as the distention was reduced to allow the muscle fibres to contract.

We have found abdominal distention present in a case of amputation of the breast which was controlled by the continuous duodenal suction. The apparatus was used to control distention in a case of ruptured gall bladder in a woman 74 years of age and in a case of a ruptured pyosalpinx with free pus in the peritoneal cavity in a woman 38 years of age.

In the group of cases we have studied we have had the most gratifying results. We usually passed the duodenal tube to the point where the pyloric sphincter would be placed normally. The fluids that were given by mouth made the patient quite comfortable and also gave us the opportunity of knowing when peristalsis had been established. It might also be said that this was, in a way, a form of continuous gastric lavage. Our best results were found where fluids were given at the rate of 3 ounces per hour.

The use of the continuous duodenal suction is an intrinsic factor in the treatment of distention. Once the stress or strain on the muscular wall of the gastrointestinal tract is relieved, the other medication takes effect. In a majority of cases we have observed a return of peristalsis on the relief of the distention with no abnormal stimulation.

SUMMARY

1. Distention is primarily an atonic condition of the musculature of the gastrointestinal tract with an increased secretion of fluid from the mucosa causing more pressure from within, accentuating the atony and thus forming a vicious cycle.

2. Continuous duodenal drainage removes the succorrhea and allows the gastrointestinal walls to return to normal so that peristalsis can be produced as the result of normal stimuli.

3. Drainage, by removing the gastric and duodenal contents, prevents vomiting.

4. By the use of the apparatus described, the patient can be made comfortable by giving fluids by mouth. If the gastrointestinal tract cannot carry the fluids down, they return through the duodenal tube.

5. The return of peristalsis is ascertained by a marked increase in the fluid intake by mouth over the amount returned through the duodenal tube.

6. When central stimulation such as obtained by using pituitary extracts, is not successful in relieving the distention, the effects can be enhanced by using continuous duodenal suction until the walls of the gastrointestinal tract return to normal.

REFERENCES

Bassler. Diseases of the Digestive System. S. A. Davis Co.

Alvarez. Mechanics of Digestion. Paul Hoeber Co.

Rehfus. Diseases of the Stomach. Phila. W. B. Saunders Co.

Babcock. Textbook of Surgery. W. B. Saunders Co.

Cecil. Textbook of Medicine. W. B. Saunders Co.

Wright, Samson. Applied Physiology. Oxford Press.

RECTAL EVIPAL AS A COMPLETE ANESTHESIA*

CLINICAL OBSERVATIONS ON 200 CASES
A PRELIMINARY REPORT

HOWARD HOGAN, M.B.

Anesthetist to the Swedish Hospital, Brooklyn, N. Y.; Associate Anesthetist to the Willard Parker Hospital

NEW YORK CITY

WE feel that the rationale of evipal sodium has been established clinically by the reports of many thousands of cases from the continent of Europe,[1] and of 25,000 cases from the Research Council of Great Britain.[2] Since 1934, many thousand cases of the successful use of evipal as an intravenous anesthetic have been reported in this country.[3]

The use of evipal sodium by rectal administration was first conceived by Gwathmey.[4] Evipal sodium is a cousin rather than a member of the immediate family of the barbituric acid group. We found it to be a mild respiratory depressant but that the pulse and blood pressure were only slightly, if at all, affected. We found it to be more anesthetic than sedative.

After experimental work in the laboratory on dogs, and in small but ever-increasing doses on human beings, Gwathmey[4] recommended an index of 0.2 times the body weight as a safe preanesthetic medication.

The drug for use rectally is distributed in 3 gram bottles containing a white, crystalline powder. The powder is shaken into 30 cc.'s of warm tap water and allowed to dissolve. A 10 per cent solution, clear, somewhat soapy to the touch, and about the same viscosity as water, results. This solution is non-irritating and causes no unpleasant sensation whatsoever when injected. This 10 per cent solution is used in all cases and doses are calculated in cc.'s. Gwathmey thus recommended a dose of 30 cc.'s for a 150 pound man as the maximum to be given. (150 lbs. × index 0.2 = 30 cc. 10 per cent solution.) In elderly, obese or debilitated patients, the dose should be reduced to slightly less than weight in lbs. × index 0.2 (20 to 25 cc.'s, etc.), while in athletic types, alcoholics or excitable patients, slightly more of the drug could be administered so long as 30 cc.'s of the 10 per cent solution was not exceeded.

I had used evipal sodium intravenously in 1935 in a series of 125 cases with very satisfactory results. At that time, we used an index of 0.06 times the body weight 150 lbs. × index 0.06 = 9 cc. of a 10 per cent solution) to obtain the dose to be administered. These amounts were added to or subtracted from, to suit the individual patient, using as a guide the general condition, type of operation, and the reaction upon administration. It seemed logical to us, then, that with the index increased for rectal administration (slightly more than three times that for intravenous use) the same rules should apply. Accordingly, we began this series, observations on 200 cases of which are reported here, with Gwathmey's index of 0.2. We added or subtracted small amounts of evipal soluble, guided by the general condition of the patient, his reaction to the pre-anesthetic medication and the type of operation. We found a useful guide in the reaction of the patient to dilaudid hydrochloride, which we use routinely in preference to morphine sulphate as a pre-anesthetic and post-operative narcotic. In patients who were made drowsy by the dilaudid hydrochloride, we reduced the amount of evipal that we had intended using and, conversely, in patients who were apparently unaffected

* A paper outlining this work and including some of these cases was read at a meeting of the American Society of Anesthetists, at the New York Academy of Medicine, February 11, 1937.

by the dilaudid hydrochloride, we increased the pre-conceived dose by a few cc.'s. Our results differed from those obtained by Gwathmey in that we found we did not have to give supplementary anesthesia terminally but at the beginning of operation. Our patients were perfectly relaxed at the end of operation after very small amounts of supplementary oxygen and ether. We felt that the reason for this difference was insufficient time allowance for the descent of the patient along the planes of anesthesia. Accordingly, we tried various times of administration from ten minutes to two hours before operation, and we have obtained the greatest effect from the drug when it is administered one and a quarter hours before scheduled operation.

The pre-anesthetic medication used is dilaudid hydrochloride gr. $\frac{1}{48}$ two hours before operation and repeated one and a half hours before. We find with this drug, in preference to morphine sulphate, that there is a reduction in post-operative nausea and vomiting from 20 per cent to 5 per cent, reduction in occurrence and severity of gas pains, and reduction in number of catheterizations. Post-operatively, we prescribe the drug in doses of gr. $\frac{1}{24}$ hypodermically every four hours as necessary.

TECHNIQUE

The evening before operation, the patient is given a light supper and at 9 P.M. a soap suds enema. If patient requires a sedative, two allonal tablets are prescribed —we believe that the more powerful barbituric acid derivatives are definitely contraindicated. On them orning of operation, a soda bicarbonate enema is given at least three hours before operation.

For a patient scheduled for operation at 10 A.M. the following procedure would be carried out: at 8:00, dilaudid hydrochloride gr. $\frac{1}{48}$ by hypodermic injection; 8:30, dilaudid gr. $\frac{1}{48}$ by hypodermic injection; 8:45, patient placed in Sims' position, small catheter introduced 3 to 4 inches into rectum and evipal 10 per cent solution injected rapidly with 50 cc. syringe; catheter is clamped and syringe removed; at 10, 15 cc. warm tap water is drawn into syringe which is reinserted into catheter; catheter unclamped and tap water is forced through catheter to clear it of any remaining evipal solution; catheter is reclamped and left in situ. The patient is then covered up and allowed to go to sleep. When called for by the operating room orderlies, he is transferred to the stretcher still in Sims' position, being placed on his back only when he is transferred to the operating table in the anesthetic room when he is in the hands of the anesthetist. The patient is left in Sims' position to avoid the danger of the tongue falling back in the throat. The drug is administered by nurses, who are instructed about colour, pulse, respiration, and the importance of the clear airway. The nurse does not give an anesthetic but merely a small retention enema, since anesthesia is not fully established until the patient arrives in the operating room when he is in the hands of the anesthetist. At this time, an airway is usually introduced and oxygen may be given, though we have not found this necessary.

No cases have given alarm; one was sensitive to dilaudid hydrochloride and one was sensitive to the barbiturates. The first was given caffeine sodio-benzoate gr. viiss every hour and carbon dioxide and oxygen mixture. The operation proceeded without event and the patient was fully conscious five hours later, reacting quietly. The second case made no sign until twenty minutes post-administration when her respirations fell to 4 and then to 2 per minute, the pulse remaining at 72. Metrazol 6 cc.'s were given in the vein and carbon dioxide and oxygen were administered. The respirations immediately rose to 6, then to 8 and at operation were 12. The colour remained good throughout, as did the pulse rate and volume. The operation proceeded normally, and three days later the patient developed an allergic skin eruption which disappeared upon the administration of Miii of $\frac{1}{1000}$ solution of epinephrine.

Post-operative patients are turned from side to side and supported by pillows every hour in order to promote bronchial drainage and to aid the circulation at the bases of the lungs. Post-operative aeration of the bases of the lungs by means of stimulation with carbon dioxide is not advocated. We feel that metrazol is a milder, more uniform, more effective means of accomplishing complete aeration of the lung than the use by the individual of the carbon dioxide-oxygen mixture with the attendant dangers of over-stimulation and laceration of the smaller alveoli. Metrazol, in addition to its action on the respiratory center which increases the depth, but not the rate of the respiratory cycle, improves the volume of the pulse. At the end of the operation, we give metrazol 3 cc. in the muscle and 3 cc. in the vein routinely. If the patient shows any sign of depression the 6 cc. are given slowly in the vein. Before the injection into the vein is completed, the excursion of the thorax is increased. When the patient is returned to bed metrazol 2 cc. are given half hourly for two or three doses. The use of metrazol has been inaugurated routinely in this series to attempt to elevate the patient to the first stage of anesthesia rather than to overcome any apparent depression. We try to restore the reflexes without wakening the patient, and in this way allow him to sleep lightly through the pain zone using the anesthetic drug as a post-operative analgesic. In this way we have been able to reduce the amount of post-operative narcotics to two or three injections per patient. The first reaction of the patient on awakening is not pain—rather they ask the time, or their whereabouts, or when the operation is to take place. Some react restlessly, but a prompt injection of dilaudid hydrochloride controls the restlessness, and the patient returns to sleep. The second awakening is similar to that from a natural sleep and a patient anesthetised with this drug, throughout the operative procedure, resembles a person enjoying a natural, restful slumber.

DOSAGE

It cannot be too strongly emphasized that no set dose can be calculated, all of which must be given previous to operation, which will be correct in all cases without approaching extreme danger. We have done laparotomies with perfect relaxation on an index of 0.26 × the body weight, and we have had to use supplementary anesthesia with an index of 0.4 times the body weight. After seventy-five years' experience with ether no one is able to tell with a very high degree of accuracy the amount which will be necessary to relax any individual patient. It is true that there is no reason to forecast the amount of an inhalation anesthetic, since the ease of addition makes the attempt at prophecy unnecessary: but with an anesthetic whose dose must be preconceived, a high degree of accuracy is necessary for its efficient use. Using rectal evipal as a complete anesthesia, we have found the following indices to be 90 per cent correct: (1) 0.2 to 0.25 for orthopedic work and for plastic operations on the vagina; (2) 0.28 to 0.34 for extra abdominal cases which includes hernias; (3) 0.35 to 0.4 for intra-abdominal cases.

In other words, we have used an index of 0.2 to 0.4 times the body weight, depending on the condition of the patient, his reaction to the pre-anesthetic medication and the type of operation. With these indices the necessity for supplementary anesthesia is rare and the amounts negligible. Should the occasion arise, we advocate the use of ether, or vinethene, with oxygen by means of the carbon-dioxide absorption technique with a vaporizer.

The safety of the drug itself seems to us to have been established by the enormous number of cases upon which it has been used as an intravenous anesthetic. In the laboratory, with dogs, the lethal dose was found to be more than four times the anesthetic dose; with ether the lethal dose was 2.5 times the anesthetic dose. The factor of safety then is 4+ with rectal evipal whereas with ether, our safest an-

esthetic until now, the factor of safety is 2.5. We have used 0.4 as our highest index, twice the basal dose recommended by Gwathmey[4] and as no higher index is necessary we would seem to have a wide margin of safety. The margin of safety is added to in that by means of the continued intravenous administration of metrazol the anesthetic can be reversed. We have awakened patients from deep anesthesia on the table by the administration of, in one case, 15 cc.'s of metrazol. Post-operatively, we find that 2 cc.'s of metrazol repeated half hourly for two or three doses keeps the patients in the desired state of analgesia, once it has been attained by the preliminary injection of 6 cc.'s at the conclusion of operation.

Post-operative patients are given clear fluids as soon as they are desired. We find regurgitation without nausea in 5 per cent of patients; there is no true vomiting with preceding nausea. Patients who do regurgitate do so once or twice, usually about twelve hours post-operative, and resume the taking of fluids at once. Twenty-four hours post-operative the patient looks and feels well, is taking and retaining fluids, has voided, has some discomfort in the operative area but no great pain. Post-operative pulmonary complications have occurred in six cases—three of whom had bronchial pathology on admission and do not properly belong in this group. Of the other three, the fourth, fifth, and sixth cases displayed evidence of "pneumonitis," ran one and two day temperatures of 101° to 102° (R) and made uneventful recoveries. This gives a percentage of 3 per cent.

One of the most encouraging results from the use of this anesthetic agent is the pleasure of the patients in having escaped the horrors of the "mask," the "whirling of black pools," and the "roaring in the ears" which perhaps cannot be measured clinically but must be of enormous psychological and physiological import.

The cases in this series vary from a boy of 3 to a venerable lady of 79, and from 40 to 260 pounds; 80 per cent were private patients, one of whom was the wife of one of the operating surgeons using this anesthetic. In the group have been 3 asthmatics, 5 diabetics, 1 aortic regurgitation with sickle celled anemia, 1 acute appendicitis with influenza, 1 acute mastoiditis with acute hemorrhogic nephritis, 1 jaundiced cholecystectomy, 1 ruptured gangrenous gall-bladder (removed), 1 ruptured ectopic who lost 1000 cc. of blood and was transfused before leaving the operating room, 2 ruptured gastric and 1 ruptured duodenal ulcers, all of whom made uneventful recoveries. These cases were obviously from the "poor risk" group. We found only slight variations in blood chemistry in these and other patients which agrees with the findings with the intravenous use of the drug.[5]

Case 1 (Fig. 1). An adult white female, aged 58, enters hospital for the repair of her pelvic floor. She is a diabetic of five years standing but it has never been necessary for her to take insulin.

Clinical examination reveals a BP of 190/110, generalized arterio-sclerosis, complete prolapse of the uterus, rectocele, cystocele.

Laboratory examination reveals a blood sugar of 210 mgm per 100 cc., 2.5 per cent sugar in the urine with a trace of albumin, many epithelial and red cells, a few leucocytes.

Patient was put on a diet and in ten days her urine was sugar free and her general condition had improved. She was scheduled for operation on the twelfth hospital day. She weighed 160 pounds and the index decided upon was 0.18 (160 × .18.) We gave her a dose of 30 cc.'s of evipal. Pre-anesthetic medication was dilaudid hydrochloride gr. $\frac{1}{20}$ given at 8:30 A.M.; the evipal was given at 9:00 A.M. for operation at 10:00. Anesthesia was established at 9:30. The operation began at 10:00 when the pulse, respirations and blood pressure were 8^2, 14, 160/100. The pulse and respirations remained the same until the repair of the rectocele, when they became 86 and 18 respectively. The patient made no protest at any time during the procedure which took one hour and twenty-five minutes. The cervix was amputated, the uterus was suspended from below and a posterior repair of the perineum was done. Patient was returned to bed at

11:40, with pulse 96, respiration 16. Metrazol 1 cc. at 11:45 and 12:45. Patient reacted at 3:00 P.M. restlessly and was given dilaudid

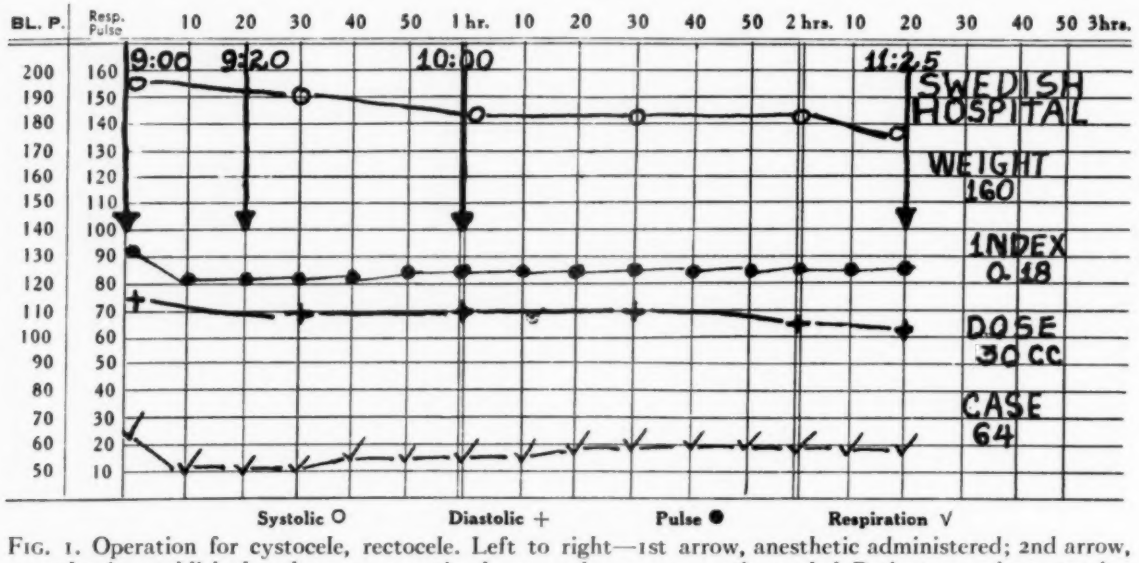


FIG. 1. Operation for cystocele, rectocele. Left to right—1st arrow, anesthetic administered; 2nd arrow, analgesia established; 3rd arrow, operation begun; 4th arrow, operation ended. Patient was a hypertensive diabetic female aged 58.

hydrochloride gr. $\frac{1}{20}$; she rested quietly thereafter. The first post-operative specimen showed a trace of sugar; next morning the specimen was negative. Patient had no nausea or vomiting at any time and for the remainder of her stay in hospital, her urine was sugar free. She was discharged in fourteen days after an uneventful recovery.

CASE II (Fig. 2). This is an experiment we performed with the author as the patient. The highest index that we had used to date was 0.275. We determined to use 0.375 × 200 lbs. and this gave a dose of 75 cc.'s.

The anesthetic was administered at 2:00 P.M. Pulse, respirations and blood pressure at this time were 100, 18, 110/80. The epival was administered and the observations made by Dr. Scotti, the house surgeon. Anesthesia was established at 2:15 and at 2:25 an airway was

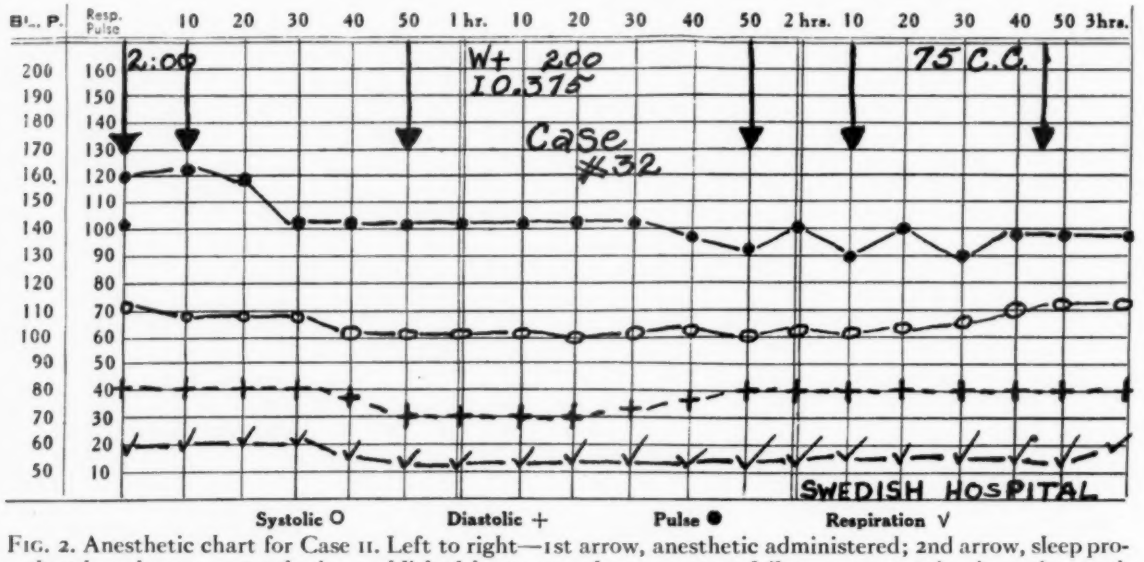


FIG. 2. Anesthetic chart for Case II. Left to right—1st arrow, anesthetic administered; 2nd arrow, sleep produced; 3rd arrow, anesthesia established by tests; 4th arrow, tests fail to cause reaction in patient; 5th arrow, patient beginning to react to stimuli. Patient was a 200 pound white adult male apparently in good condition.

introduced because the tongue had receded into the throat. At 2:50 there was no response on (a) pin sticking (b) kneading upon abdomen

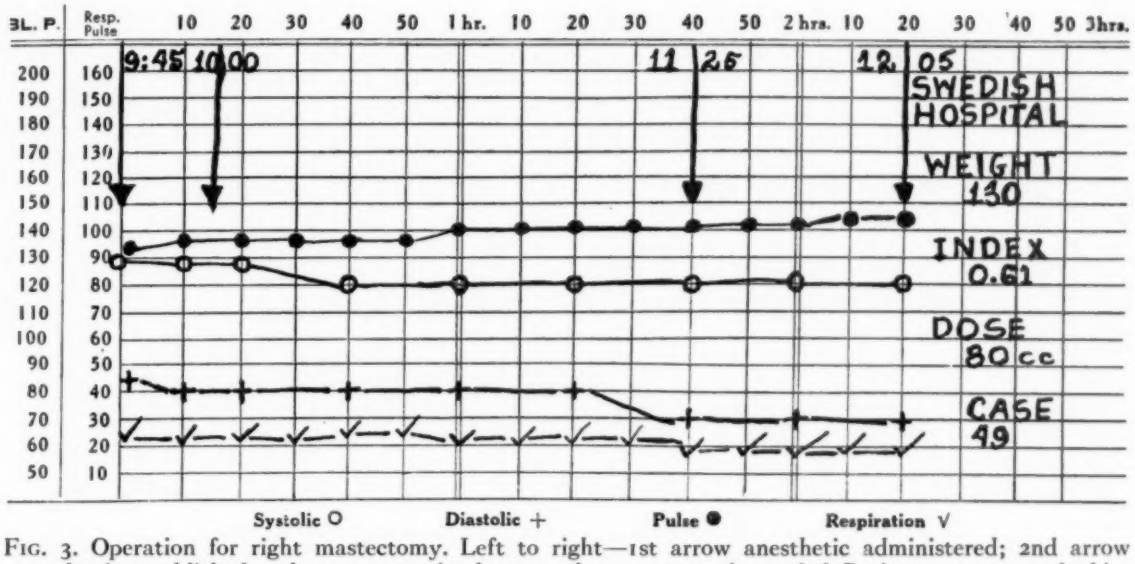


Fig. 3. Operation for right mastectomy. Left to right—1st arrow anesthetic administered; 2nd arrow analgesia established; 3rd arrow operation begun; 4th arrow operation ended. Patient was a normal white female aged 42.

formed we cannot say. We felt justified in continuing slowly to increase the index in appropriate cases.

(c) ringing of loud alarm clock in ear (d) firm pressure on supra-orbital notch (e) pinching abdomen with hemostat. Over an hour later, there was still no response to these stimuli; the eyes were slightly dilated. Pulse was 90, respiration 14, blood pressure, 98/70. At 4:10 patient began to respond to stimuli and at 4:20 responded more actively with movements of the head. Knee jerks are present and patient takes a deep gasping breath when stimulated in this manner. At 5:05, airway was removed, 5:40, eyes were open and at 6:00, patient apparently recognized Dr. Scotti.

Complete restoration of memory occurred at 9:30 and the sensation at that time was "shakiness." Dilaudid hydrochloride gr. $\frac{1}{20}$ was given and patient slept quietly until 3:00 A.M. when dilaudid hydrochloride gr. $\frac{1}{24}$ was given. Patient arose at 7:30 A.M., shaved, and carried on routine duties. The patient admits that he was fairly drowsy over the week-end but suffered no ill effects from the anesthetic.

The lack of a surgical procedure detracts from this experiment, but in the opinion of several people who observed it, there would have been no difficulty in carrying out any surgical procedure at any time without protest from the patient. Whether or not shock would have supervened had an operation been per-

Case III (Fig. 3). Adult white female, aged 42, who enters for the removal of the right breast.

The patient weighed 130 pounds and the index used was to be 0.28 (130×0.28). We gave a dose of 36 cc.'s, but owing to a mistake she was given also the dose intended for another patient, an additional 44 cc.'s. In all, this patient received 80 cc.'s. The preanesthetic medication was dilaudid hydrochloride gr. $\frac{1}{32}$ at 9:15, the evipal at 9:45. Owing to the schedule being delayed this patient did not come to operation until 11:25. At this time the pulse, respiration and blood pressure were 96, 18 and 120/70. At the time of administration these values were 90, 24, 110/70.

The incision was made and the operation proceeded without any apparent distress and ended at 12:03 with the pulse, respiration, blood pressure readings 104, 18, 120/70. The patient was returned to bed at 12:10 with the pulse 90, respirations 20, and blood pressure 116/72. Metrazol 1 cc. was given every hour and 50 cc. of 50 per cent glucose was given intravenously at 3:00 P.M. The patient began to react at 7:00 and at that time had some water. During the night she vomited small amounts of green fluid twice. At 11:30 P.M. the pulse was 108, respiration 16—the same values as had been obtained at 8:00 P.M.

The next day patient was restless and somewhat drowsy but otherwise showed no signs of her massive dose of evipal. No further nausea or vomiting occurred, her recovery was uneventful and she left hospital on the tenth post-operative day.

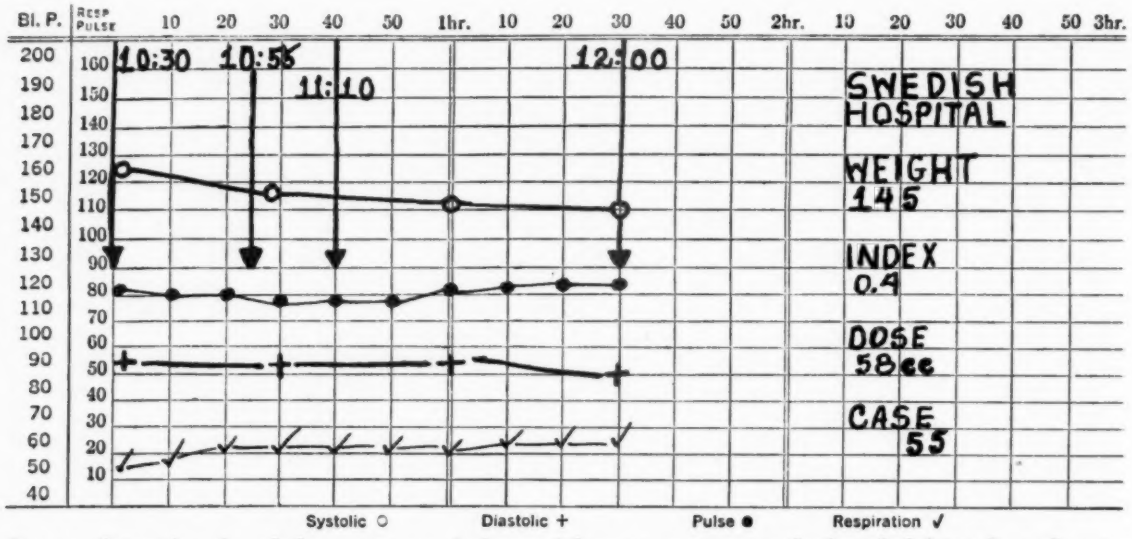


FIG. 4. Operation for cholecystectomy. Left to right—1st arrow anesthesia administered; 2nd arrow analgesia established; 3rd arrow operation begun; 4th arrow operation ended. Normal white female aged 52.

The index in this case was 0.61 × 130 lbs.—a dose of 80 cc.'s.

CASE IV (Fig. 4). Adult white female, aged 52, who enters for the removal of her gall-bladder.

Clinical examination reveals pain and tenderness over the gall-bladder region, otherwise essentially negative.

X-ray examination shows a pathological gall-bladder with calculi.

Patient weighed 145, the index was 0.4 (145 × 0.4) and the dose of evipal 58 cc.'s.

Pre-anesthetic medication dilaudid hydrochloride gr. $\frac{1}{24}$ at 9:30 A.M., dilaudid hydrochloride gr. $\frac{1}{32}$ at 10:00 A.M. and rectal evipal at 10:30 A.M. for operation at 11:00. Pulse 72, respiration 20, blood pressure 162/94 at the time of administration. Anesthesia was established at 10:55.

The operation began at 11:10 and lasted fifty minutes. The gall-bladder was removed. The only sign given by the patient during the procedure was a deep gasping breath when the gall-bladder was tugged upon. The abdomen was closed without difficulty at 12:00. Patient returned from operating room at 12:20, pulse 88, respiration 16 and blood pressure 150/90. Metrazol 1 cc. at 12:30, 1:30, 2:30. Reacted at 3:20 when dilaudid hydrochloride gr. $\frac{1}{24}$ was given and patient rested quietly thereafter. At this time pulse was 100 respiration 18.

From 4 A.M. the next day, patient drank all fluids including beer that afternoon. She had bouts of regurgitation but her post-operative course was uneventful and extremely satisfactory.

This is the first case in which we have divided the dose of dilaudid hydrochloride for increased analgesic effect. The patient was almost asleep from the effects of the pre-anesthetic medication when the evipal was administered.

SUMMARY OF CASES*

General Surgery

Amputations—thumb 1, finger 1, leg 2	4
Appendectomies	52
Cholecystectomies	10
Curettage of tibia	1
Cellulitis of groin (incision and drainage)	4
Cellulitis of neck (incision and drainage)	1
Colectomy (partial)	3
Duodenal ulcer (perforated) closed	1
Excision lipoma of buttock	1
Foreign body in hand 1, leg 1	2
Gastric ulcer (perforated) closed	2
Haemorrhoidectomy	9
Hernia, bi-lateral 3, uni-lateral 8	11
Hydrocele	2
Mastectomy bi-lateral 1, uni-lateral 11	12
Pilonidal Cystectomy	4
Thyroidectomy	1
Thoracotomy	1
Thyro-Glossal cyst	1

Genito-Urinary Surgery

Nephrectomy	1

Amputation of penis	1
Urethro vaginal fistula (repair)	1
Orchidectomy	1
Gynecology	
Amputation of cervix	6
Cystocele	11
Dilatation and Curettage	4
Ectopic gestation	1
Hysterectomy	6
Myomectomy	1
Oophorectomy	6
Rectocele	11
Salpingectomy	6
Uterine suspension	4
Uterine polyp	1
Obstetrics	
Caesarian section	3
Analgesia in normal delivery	3
Orthopedics	
Reduction fractured metacarpal 1, elbow 1, colles fracture 2, humerus 1, femur 2, patella (open) 1, tibia and fibula 1, potts 1	10
Ear, Nose and Throat	
Mastoidectomy	4

* Each of the above procedures represents an individual patient.

We cannot emphasize too strongly the necessity of experience with patients under general surgical anesthesia. A knife is dangerous in the hands of a child, a scalpel is life-saving and life-giving in the hands of a skilled surgeon. All anesthetics are dangerous in the hands of those not trained in their use. Evipal soluble, used rectally, has proved in our hands to have a definite place in the armamentarium of surgery and its allied sciences.

SUMMARY

1. Clinical observations of 200 cases of rectal evipal as a complete anesthesia are reported.

2. Depth of anesthesia is easily controlled by means of metrazol.

3. Post-operative pulmonary complications have been markedly reduced in this hospital since the introduction of the described Metrazol technique.

4. Post-operative distress has decreased and hospitalization of patient has been reduced by one to three days.

5. In some cases, the relaxation in the abdomen has not been satisfactory and it is our belief that the addition of a small amount of ether in oil to the rectal evipal will eliminate this factor. Clinical observations along these lines are proceeding.

REFERENCES

1. Reference to many thousand cases in Europe.[3] Killian, Hans. "The New German Evipan-Sodium Anaesthesia." Current Researches *Anaest. & Anal.*, 13: 177–183, 227–232, 1934. Wesse, H. *Deutsche med. Wchnschr.*, 58: 1205–1207, 1932; 59: 47, 1933; *Fortschr. d. Therap.*, 8: 487, 1934.

2. Report of Anaesthetic Committee of the Medical Research Council of Great Britain.[4] *Lancet*, 5731: 43, 1933.

3. Many cases in this country: Veal and Hamilton. *Anaest. & Anal.* (Sept.-Oct.) 1936. Livingston, Emy, Leiber. "Evipal Soluble, A Short Intravenous Anaesthesia." *Am. J. Surg.*, 26: 516–521, 1934, No. 3. Suraci, Charles X. "Evipal Soluble, A Unique Anaesthetic for Intravenous Use." *Med. Ann. Dist. of Columbia*, 4: 29 (February) 1935.

4. Gwathmey, James T. "Rectal Administration of Evipal Soluble—A Preliminary Report." *Am. J. Surg.*, 33: No. 3; 411–416, 1936.

5. Veal, J. R. and Hamilton, A. S. Further Observations on Evipal Anesthesia. *Anaest. & Anal.*, 15: 231–236 (September-October) 1936.

ANTISEPTICS AND WOUND HEALING*

W. Warren Sager, M.D., Edward B. Vedder, M.D. and Charles Rosenberg, M.A.

Associate in Surgery — Professor of Experimental Medicine — Research Assistant in Experimental Medicine

George Washington University Medical School

WASHINGTON, D. C.

THE history of the treatment of wounds is as old as the history of the human race. Unfortunately lacking the knowledge Pasteur gave us of bacteriology, the treatment given was frequently ineffective and at times cruel. The knowledge that wounds will heal if accurately approximated may have been known to some ancient surgeons, and Henri de Mondeville (1260–1320) gave some excellent directions for the aseptic treatment of wounds. But these suggestions were not followed by medieval practitioners, whose efforts were concerned with keeping the wound open to allow for the drainage of laudable pus. This of course, was justified in some wounds.

Paré's observation in 1536 that wounds treated with a "digestive of yolkes of eggs, oil of roses and turpentine" did better than those into which he poured his burning oil was a great discovery, for it had been supposed that all gunshot injuries were poisoned.

John Hunter in the latter part of the eighteenth century taught the surgeons of his day to return to simple dressings and allow Nature to heal in her own way. Unfortunately following this period the Napoleonic wars introduced a great number of patients with severe and badly infected wounds which completely changed the type of surgery taught by Hunter. At this time the mortality after major amputations in civil hospitals varied from 20 to 60 per cent, as quoted by Ashhurst.

Such was the surgical world into which Lister introduced antiseptic surgery after his attention had been drawn to the work of Pasteur. Lister when operating kept his marine sponges and instruments in a carbolic solution, washed his hands and painted the part to be operated upon in the same solution. He used a carbolic spray over the wound, drapes, surgeon, assistants and visitors. Before closing, Lister rinsed his wound with a 1:40 carbolic solution. Catgut used was soaked in a 1:20 carbolic solution, and additional carbolic solution was conveyed to the wound by sponges. All wounds were drained. Consideration of Lister's method impresses the fact that except for the introduction of antiseptic into the wound itself, he was practicing aseptic surgery, crudely, but with extreme thoroughness. Had he prevented the introduction of antiseptic into the wound, drainage would not have been necessary.

Impressed by the numerous new antiseptics being used and the lack of controlled experimental data on living animals the following study was done. Many powerful antiseptics are known by their lethal effects on bacteria in vitro and in some instances, by the addition of blood, tests have been performed in such a fashion as to simulate closely the conditions in a wound. Excellent work has been done showing the relative toxicity of various antiseptics on bacteria and tissue cultures. But the one factor not adequately studied has been the effect upon the cells of the living animal. A number of studies have been made of the effects of various antiseptics on the skin. But in this study we are concerned only with the action of antiseptics in wounds as

* From the Department of Experimental Medicine, George Washington University Medical School.

opposed to skin, mucous membranes and peritoneum which all have a very definite protective covering of epithelium.

Technique. A healthy dog was anesthetized and bilateral incisions about two inches long were made in the abdominal wall and carried down to the peritoneum. Each incision was placed the same distance from the midline and both were of the same length. After careful hemostasis each wound was massively infected with an equal quantity of a bouillon culture of a recently isolated and virulent culture of Staphylococcus aureus. One side was then treated by holding it open and filling with the antiseptic which was allowed to remain for twenty to twenty-five minutes. Both wounds were then closed in layers and a collodion dressing applied. The wounds were carefully observed until healing in both was complete, any difference in appearance being noted. It will be seen that the untreated wound serves as an excellent control upon the effect of the antiseptic. The following tables give the results obtained:

TINCTURE METAPHEN

	Control Wound	*Antiseptic in Wound*
Dog 1.	Slight inflammation.	Wound open and draining pus.
Dog 2.	Slight inflammation.	Wound open and draining pus.

TINCTURE MERTHIOLATE

	Control Wound	*Antiseptic in Wound*
Dog 1.	Open, draining pus.	Open, draining pus.
Dog 2.	Healed without evidence of infection and very little inflammation.	Healed without evidence of infection and very little inflammation.
Dog 3.	Moderate inflammation; no purulent drainage.	Draining pus, wound open throughout.
Dog 4.	Healed with very slight inflammatory reaction.	Healed with very slight inflammatory reaction. An abscess developed further back on abdominal wall apparently communicating with this wound; abscess in midline.
Dog 5.	Healed with very slight inflammatory reaction.	Healed with very slight inflammatory reaction. Abscess developed in the midline which I believe communicates with this wound.

GENTIAN VIOLET 2 PER CENT AQUEOUS SOLUTION

	Control Wound	*Antiseptic in Wound*
Dog 1.	Wound healed with very slight inflammatory reaction.	Wound healed with very slight inflammatory reaction.
Dog 2.	Wound healed with very slight inflammatory reaction.	Wound open and draining pus; open throughout.
Dog 3.	Healed with very little inflammatory reaction. No drainage.	Healed with moderate inflammatory reaction; no drainage.
Dog 4.	Healed with very little inflammatory reaction.	Wound open throughout and draining pus.
Dog 5.	Healed with very little inflammatory reaction.	Healed without draining but markedly edematous and inflamed.

AZOCHLORAMID

	Control Wound	*Antiseptic in Wound*
Dog 1.	Badly infected, draining.	Badly infected, draining pus, open.
Dog 2.	Healed with slight inflammatory reaction.	Healed with slight inflammatory reaction.
Dog 3.	Markedly inflamed; slight serosanguinous drainage.	Markedly inflamed; no drainage.
Dog 4.	Healed with very slight evidence of infection.	Purulent drainage.
Dog 5.	Marked edema and swelling; no drainage.	Purulent drainage.
Dog 6.	Wound open and draining pus.	Wound open and draining pus.
Dog 7.	Wound open and draining pus.	Wound open and draining pus.
Dog 8.	Wound healed with marked inflammatory reaction.	Wound healed with marked inflammatory reaction.
Dog 9.	Healed with no evidence of infection and little inflammatory reaction.	Opened very little and drained a very small amount of pus.
Dog 10.	Purulent drainage.	Sinus on abdomen back of wound communicating with wound; purulent drainage.

CONCLUSIONS

Using the antiseptics noted in the tables we were unable to improve the condition of our infected wounds. Gentian violet with a bacteriostatic value rather than a bacteriocidal one gave about the same results

as the more powerful antiseptics. Similar results may be expected from any antiseptics now used and these results are due to injury of the cells by the antiseptic. We feel that as antiseptics are necessary and will continue to be used more knowledge of their effect upon cells and bacteria in wounds should be obtained.

REFERENCES

Ashhurst, A. P. C. Centenary of Lister (1827–1927); tale of sepsis and antisepsis. *Tr. Coll. Physicians*, 49: 70–93, 1927.

Buchsbaum, Ralph and Bloom, William. Relative toxicity of antiseptics on bacteria and tissues in cultures. *Proc. Soc. Exper. Biol. and Med.*, 28: 1060–1064, 1930–1931.

Morris, Robert T. Fifty Years a Surgeon. New York, E. P. Dutton & Co., Inc., 1935.

Billroth's disease (malignant lymphoma) is named for Theodor Billroth (1829–1894), a surgeon of Vienna.

Bright's disease was first described by Richard Bright (1789–1858) in 1827.

Calvé-Perthes disease, osteochondritis deformans juvenilis coxae, derives its name from Jacques Calvé, a French orthopedist, and Georg Clemens Perthes, a German surgeon.

Duchenne's disease, locomotor ataxia or pseudohypertrophy of muscles, is named after G. B. A. Duchenne (1806–1875), a French neurologist.

Erb's atrophy or disease, takes its name from Wilhelm Heinrich Erb, born in 1840, a physician of Heidelberg.

Graves' disease, exophthalmic goiter, is named after Robert James Graves (1797–1853), an Irish physician.

INTESTINAL RESECTION BY A SINGLE CLAMP METHOD*

N. N. SSAMARIN, M.D.

LENINGRAD, U.S.S.R.

AS is well known, the continuity of the gastrointestinal tract may be restored after resection by an end to end, end to side, or side to side union. Technically, the side to side procedure is simpler, while the end to end techinque is approximately midway between the aforementioned procedures in respect to difficulty.

However, not only the technical difficulties of the method,[1] but the physiologic demands of the desired anastomosis must be considered. In end to side and side to side union intestinal pouches may be formed which are undersirable as they may produce pain occasioned by the stagnation of intestinal contents. The end to end union never leads to such diverticuli and is thus more physiologic and consequently more desirable. Since the properly performed end to end anastomosis is the most adequate one, our problem resolves itself into working out such a procedure which would be simple and relatively safe for the patient. Bearing in mind these two main points one has to acknowledge the following principles: (1) the operation must be an aseptic one and (2) postoperative strictures, bleeding and possible rupture of the suture line are to be avoided.

Many surgeons have devised methods aiming to surmount all these difficulties. Several aseptic procedures have been proposed, among which I should like to mention the colonic resection of Schoemaker, as a rather simple one. Schoemaker proceeds as follows: After making a circular incision of the serous and muscle layers of the colon, both layers are elevated from one end of the segment to be resected. The naked tube of mucous membrane thus produced is clamped by two thin bladed stout clamps placed very near one another, and the colon is sectioned between them. Each end of the segment to be removed is then sectioned in turn. The clamps on the proximal and distal stumps of the bowel are then approximated. The serous coat is first sutured posteriorly by interrupted sutures, then the mucous muscle and serous layers. This done, the clamps are turned around their axes and anterior sutures are inserted through all three layers, the clamps being removed as the last suture on both borders are tied; lastly, an anterior suture of the serous coat is placed.

Such a resection is relatively aseptic, very convenient from the point of view of technique, and requires no special instruments. Still I believe a modification might appear desirable. The suturing might be made easier without interfering with the asepsis. With this in mind I planned the following procedure. Once the mesentery has been divided and tied, the bowel segment to be resected is mobilized and turned into a loop, in such a way that points where the end to end anastomosis has to be done are to lay one upon the other (Fig. 1). That manipulation is rather a simple one. The superimposed ends of the loop of bowel are fixed together by a suture inserted on the mesenteric border, taking care that the severed edges of the mesentery are closely approximated. Now one must choose the future line of resection, the essential point of the procedure. Kocher in his rather old textbook on operative surgery advocates the oblique section of the bowel, with a greater quantity of tissue excised from the antimesenteric border. He aims thus to prevent cutting blood vessels running transversely in the

[1] SSAMARIN, N. N. On the technic of intestinal resection. *Vestnik Kbirurgii* (Nos. 56–57) 19: 342–345, 1930.

* From the 2nd Surgical Clinic of the Kirov Post-Graduate Institute of Medicine, Leningrad, O. L.

bowel wall with consequent ischemia of a portion of the anastomosis. Besides securing the best nutrition, the oblique section also aids in obtaining a sufficiently large stoma, which is a very important feature. A transverse section with subsequent end to end suture could produce a narrowing of the lumen, a dangerous result to be carefully avoided. With this in mind, once the suture on the mesenteric border is applied, the antimesenteric borders of the bowel are approximated to each other in such a way that the future line of section may receive a minimum angle of obliquity of 30° to 40°. A suture is now inserted on the antimesenteric border to fix the bowel in the desired position.

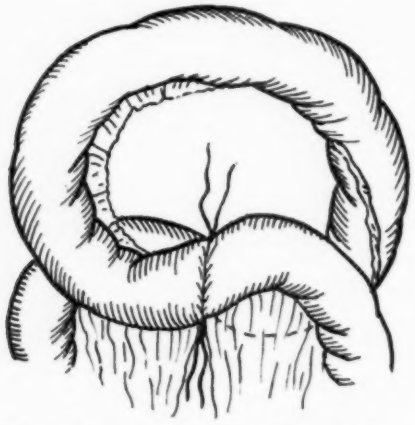


Fig. 1. The segment to be resected has been divided from its mesentery and formed into a loop. At the point of crossing of the superimposed intestinal limbs two sutures are applied, one at the mesenteric and one at the antimesenteric border. The rule of Kocher is observed, that is; an oblique line of section is planned.

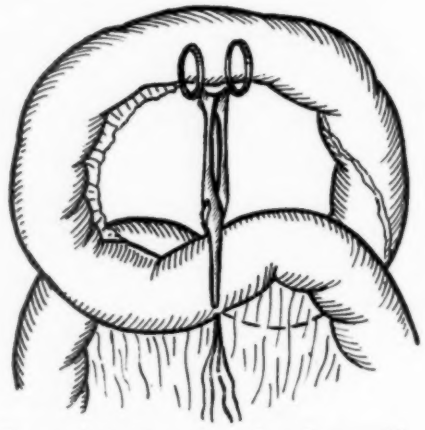


Fig. 2. A stout compressing clamp without teeth has been applied in the line of the first sutures, thus fixing the superimposed loops one to another.

This done one has to stretch gently the threads of the sutures in opposite directions in order to straighten the loops and to pass them hereafter to the assistant who is to hold them. The superimposed intestinal segments are then clamped together by one Kocher hemostat without teeth as in Figure 2. The tip of the clamp should be placed on the bowel 1 mm. from its mesenteric border. The Kocher clamp is now closed to the last lock in order to

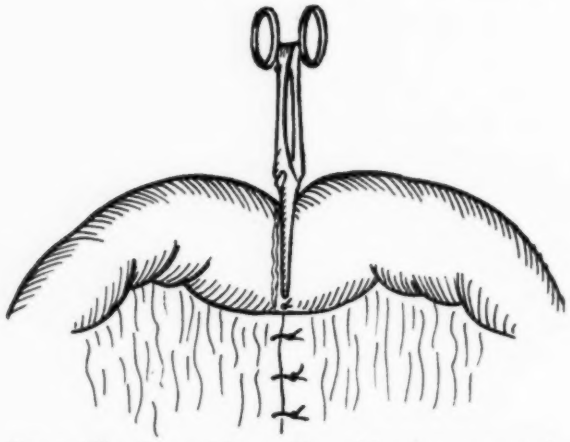


Fig. 3. The diseased loop of bowel has been resected, but the stout clamp links both stumps together. Suturing may now be begun.

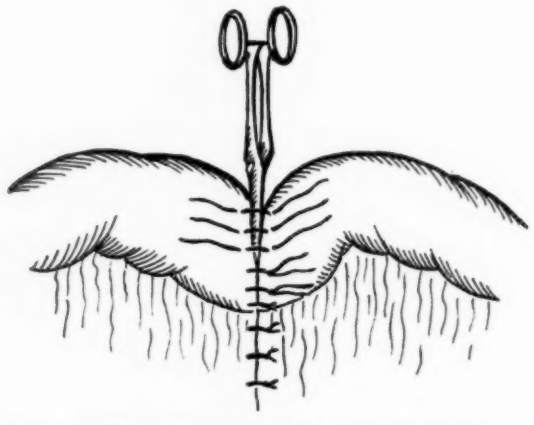


Fig. 4.—The clamp is being covered by interrupted Lembert sutures of silk.

crush the loops thoroughly. The intestinal loops are now properly fixed at the point where section is to be performed. Much care has to be taken to prevent the escape of intestinal contents from the lumen of the sectioned part of the bowel. To avoid this two clamps are fixed on this segment contiguous with the central fixing clamp. The bowel is now divided first from one side of the central clamp and then from the other (Fig. 3). Both lines of section are thoroughly cauterized with carbolic acid solution and the application of sutures is begun.* This procedure does not present any difficulty as the intestinal walls slightly dilated with intestinal contents, tend to rise up from under the clamp, and no traction is needed to approximate them. The assistant has only to tighten the stitches of the continuous suture or to tie the interrupted sutures (Fig. 4). In the Schoemaker method two clamps have to be covered which is of course more difficult than covering one clamp. The sutures are applied first on the mesenteric border where they must be carefully inserted and tied lest the silk thread incise the tissue. The row of sutures is extended to the antimesenteric border. The opposite side of the bowel is similarly treated. The last two sutures on the free intestinal border are applied but not tied, but are gently drawn taught while the clamp is withdrawn with caution. The removal of the clamp covered with sutures is com-

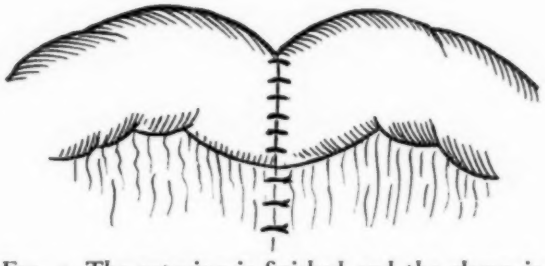


Fig. 5. The suturing is finished and the clamp is withdrawn.

Fig. 6. The invaginating finger of the operator smooths and stretches the line of union.

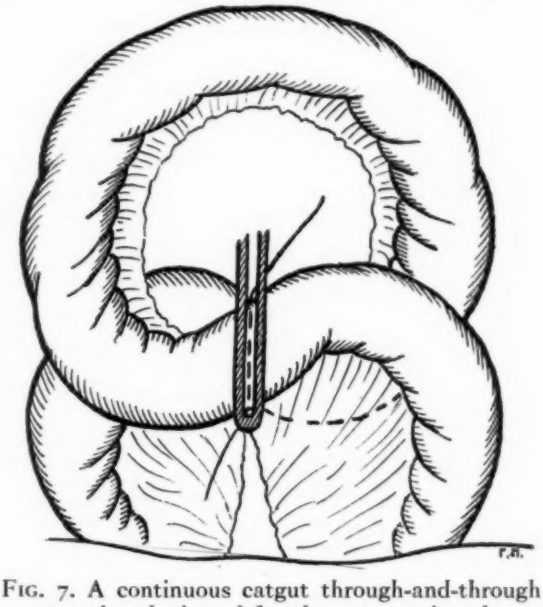


Fig. 7. A continuous catgut through-and-through suture is substituted for the compressive clamp, by inserting it through the midline opening between the blades.

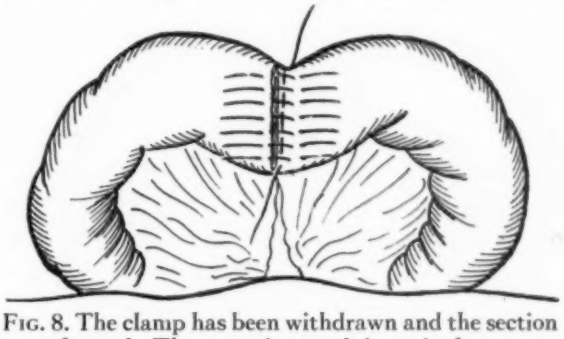


Fig. 8. The clamp has been withdrawn and the section performed. The superimposed intestinal stumps, thoroughly fixed together by a continuous catgut suture, are now permanently sutured with silk. In case of bleeding a ligature may be easily applied.

* It would be perhaps very useful to coagulate the clamped ends of the intestines in order to prevent a possible bleeding. This could be done by simply connecting the clamp with a diathermy apparatus.

paratively easy. The last two sutures are now tied and the continuity of the intestinal canal is restored by this first layer of suture (Fig. 5). The application of the second layer of sutures presents no difficulties.

The idea of using one clamp has previously been mentioned in the literature. In 1929 Horsley[2] described a single clamp procedure for the removal of a diseased segment of bowel. He used the clamp of Payr, and his technique is well demonstrated by an accompanying diagramatic sketch. The author himself criticises this method as not securing the best nutrition of the sutured ends of the bowel. But why there should be any interference with the blood supply if the vessels are not damaged is not quite comprehensible and there must be other reasons against the method.

When the suturing is done, either by Shoemaker's method or by my own with one clamp, it will be important at the last moment to restore the continuity of the intestinal lumen by gently kneading the suture line with the finger (Fig. 6).

This procedure was applied by several surgeons in this country for small intestinal and colonic resections. The data are as follows for acute and chronic cases:

	No. of Resections	Cured	Dead
N. N. Ssamarin	6	4	2
I. S. Lindenbaum (clinic of Prof. Hesse)	6	5	1
D. N. Fedoroff	1	1	0
A. P. Nadein	2	2	0
G. D. Obraztsoff	1	1	0
W. M. Woskresensky	1	1	0
P. S. Federoff	1	0	1
	18	14	4

The small number of cases precludes the possibility of drawing statistical conclusions, but a detailed analysis of some of the cases may be of use in pointing out certain preventable complications.

In one case the patient died from a secondary emergency operation to relieve partial obstruction at the first end to end anastomosis. This had occurred because of a too great infolding of the sutured borders.

Another case, operated by the author, died thirty-six days postoperatively from rupture of the suture line at the mesenteric border.

In a third patient gross rectal bleeding occurred following resection of the sigmoid. This was insignificant, however, and the patient recovered.

These complications were the only ones attributable to the method itself. The question arises, How best to avoid them? I still believe that the described method of superimposing the limbs of the intestinal loop to be resected gives the best opportunity to obtain a thoroughly made suture line, but we may dispense with the clamp in order (1) that it may not interfere with the application of the suture, (2) that a shorter end of the intestinal stump need be infolded, and (3) that bleeding may be controlled under direct vision.

For these reasons I proposed to my collaborator, Dr. G. A. Gomzjakoff, that he work out the following modification on the cadaver.

The procedure is the same to the moment of applying the clamp. The superimposed intestinal limbs are clamped, however, not with a Kocher clamp, but by a clamp used for intestinal resection with a longitudinal opening in the midline. The superimposed limbs are now temporarily sutured together by a through and through continuous catgut suture (Fig. 7), thus closing the lumen. The clamp is then withdrawn. The clamps are applied on the loop to be resected in the usual manner, but the section line is now placed at 1.5 to 2 mm. from the catgut suture (Fig. 8). Bleeding vessels may now be ligated if necessary. The suturing of the bowel over the catgut suture being completed this temporary gut

[2] Horsley. *J. A. M. A.*, 93: 1373, 1929.

suture is cut from one end and withdrawn from under the permanent sutures by traction in the opposite direction.

The only drawback to this method is the slight complication of the procedure by the necessary temporary suture. The advantages, already mentioned in part are: (1) the narrowness of the intestinal cuff which it is necessary to infold, (2) the lack of encroachment on the lumen, (3) adequate hemostasis, and (4) a very reliable anastamosis of the superimposed limbs of the loop because of the freedom of the operator to make stitches in the needed quantity and direction.

This last modification may be compared with the intestinal resection proposed by Kerr[3] who closed both intestinal ends by a basting stitch, and withdrew them once the permanent sutures were applied.

SUMMARY

Both the herein described modifications of terminal enteroenterostomy utilize a method of suturing the limbs of an intestinal loop previously fixed to one another by a clamp or by a suture. Such a method is advantageous because it may be rapidly and easily accomplished, and allows for a tight anastomosis by a completely aseptic procedure. Other points of technique are identical to all other described methods of terminal union. The two described innovations are, however, worth consideration among other proposed methods.

[3] Kerr. *J. A. M. A.*, 81: 641, 1923.

The possibility of cure of cancer of the oesophagus depends to some extent on the location of the growth. In the accessible cervical portion a number of cures have been recorded by different methods, while in the thoracic part complete cure is very rare.

From—"Diseases of the Nose, Throat and Ear" by I. Simson Hall, M.B., CH.B., F.R.C.S.E.

CASE REPORTS

PRIMARY OMENTAL TORSION

REVIEW OF LITERATURE AND REPORT OF TWO CASES

EDWARD K. BARSKY, M.D. AND ALBERT M. SCHWARTZ, M.D.

Associate Surgeon, Beth Israel Hospital

Assistant Surgeon, Beth Israel Hospital

NEW YORK CITY

INTRODUCTION

AN unexpected operative finding prompted us to investigate the subject of primary omental torsion. The infrequency of this condition, its diagnostic difficulties, etiological factors and mechanical and physical features form an interesting basis for study. Our enthusiasm was further stimulated when we had occasion to operate upon what proved to be the youngest case of primary omental torsion on record. The paucity of reports in the literature, the addition of two cases, one in a child of five, and various features of this condition seem to us to be sufficient reasons for this paper.

HISTORICAL

According to Jeffries, the first mention of torsion of the omentum was made by Pierre de Marchettes in 1851 without, however, any differentiation between primary or secondary types. In 1882, Oberst published the first complete operative description in which a band was found adherent to an inguinal ring. Up to 1931 there were reported twenty-two cases of primary omental torsion. During the same period one hundred and forty-seven cases had been recorded of the secondary type in which the presence of a hernia was the distinct etiological factor. Benton quotes one case of primary torsion reported by Eitel in 1899. McWhorter in 1928 collected twenty-four such cases, and eighteen others have been described up to 1933. D'Errico, in 1930, gathered twenty-nine cases in a study of the literature of 1899 to 1928. In 1933, Allen making a complete study found only forty-one cases of primary omental torsion. Since that publication, we have been able to locate nineteen more, and are now adding two, making a total of sixty-two cases.

DEFINITION AND CLASSIFICATION

By torsion we mean a rotation of the omentum on its axis sufficient to produce disturbances of the circulation. The rotation is usually on the longitudinal axis, though occasionally some other axis may be involved. D'Errico defines torsion as a "twisting upon itself of an organ or growth with strangulation of tissue." The ovary is the organ most frequently involved; the omentum is the least frequent offender.

Many contributors to this subject have advanced various classifications. Morris describes two types: unipolar, with one edge attached and one edge free; bipolar, with two attached points. The latter is the common variety in which the omentum had become adherent within a hernial sac or to a diseased organ. Jeffries speaks of three types: one without hernia or other apparent cause; hernial type; a type associated with intra-abdominal pathology, such as, tumors, cysts, etc., of the omentum or

* From the Surgical Service of Dr. H. E. Isaacs, Beth Israel Hospital, New York City.

other pelvic or abdominal disease. Cowell offers the following classification:

1. Abdominal
    a. No apparent cause
    b. Associated with disease of abdominal or pelvic viscera
2. Hernial
    a. Intrasaccular
    b. Intra-abdominal
    c. Combined

Certain authors write of idiopathic torsion, indicating those cases not definitely associated with hernia or any other apparent cause within the abdomen. It seems to us that simplicity and completeness could be combined in the following classification:

1. Primary—no apparent cause
2. Secondary—associated with herniae, tumors, cysts, etc., of the omentum or other intra-abdominal pathology.

Other groupings, such as pure abdominal, or pure intra-abdominal, acute and chronic, complete and incomplete, are confusing and certainly non-descriptive.

ANATOMY

The stomach appears in embryos of 4 mm. to 5 mm. as a laterally flattened fusiform enlargement of the foregut, attached dorsally to the body wall by its mesentery which later becomes the greater omentum, and ventrally to the liver by the lesser omentum. The stomach then undergoes a series of changes which result in the dorsal or greater curvature lying to the left, and the ventral or lesser curvature to the right. When the dorsal wall of the stomach rotates to the left, the greater omentum is carried with it. During fetal life the greater omentum grows rapidly to the left and caudad, in the form of a sac, flattened dorso-ventrally. It overlies the intestines and contains the inferior recess of the omental bursa. In the fourth month the dorsal wall of the sac fuses with the transverse colon and mesocolon where it lies over them. Caudal to this attachment the walls of the bursa fuse and in the adult no cavity can usually be demonstrated.

Upon opening the abdomen in the adult, there is seen extending downwards from the greater curvature of the stomach a broad, sheet-like fold of peritoneal membrane, called the greater omentum. It usually contains a quantity of fat in its meshes, and is spread out like an apron covering the abdominal viscera. Sometimes the omentum is short and narrow or it may be turned completely upwards or to one side. The more common anatomical varieties will be further discussed under etiology.

ETIOLOGY

The etiology of primary omental torsion is obscure. Certain factors, however, have been found more or less frequently associated with the condition, and while it is difficult to demonstrate conclusively a cause and effect relationship they must be considered as contributory agents. Statistical and experimental work has been done which tend to substantiate the claims concerning these points. The pertinent causes can be classified as predisposing and exciting. By predisposing we refer to those elements which of themselves are not responsible for the torsion, but so dispose the omentum that it is more susceptible to twisting. In this group the following are worthy of attention:

Anatomical Variations: The shape of the omentum varies greatly in different individuals: in some it is a broad, square or oblong structure with more or less even borders and surfaces. In others, it may be either short or long, narrow or irregular. Many have tongue-like processes attached to their free borders, which are most commonly found on the right side opposite the attached edge. In a number of specimens the right side was found to be significantly longer than the left. Bifid omenta are by no means rare. The last three mentioned types, i.e., with tongue-like projections, longer right sides and bifid formation, are thought to be more prone to torsion than the more regular ones. Our two cases illustrate the latter varieties. Congenital accessory omenta have been found attached

to the main organ, as well as congenitally malformed organs with definite pedicle formation, both of which might very easily become involved in a pathological twist.

Obesity: The majority of reported cases of torsion have been in individuals with some degree of obesity, which has affected the omentum as well as other structures. As a result of this condition, fatty masses may accumulate irregularly in the omentum, and because of the added weight may unbalance the normal equilibrium of the organ. In the presence of an exciting cause, twisting ensues. This was demonstrated by the experimental work of Payr in 1902. He injected air and solid substances into the free borders of omenta in animals, and at subsequent operation found torsion of that organ in a significant number of cases.

Arrangement of the Blood Vessels: According to competent observers, the most important predisposing factor in torsion of the omentum is the arrangement of the blood vessels. The veins are larger, longer and more tortuous than the arteries. If the veins become kinked due to any twisting force, they immediately fill up with blood, become swollen and heavier, and rotate about the arteries which form tense axial cords. This torting motion is imparted to the rest of the omental structure and once begun, the process perpetuates itself leading ultimately to infarction and gangrene.

The exciting causes are those factors which are considered capable of initiating the twist, the eventual outcome of which depends upon the degree of torsion, the duration of the process, and the concomitant circulatory disturbances. These agents can, in the presence of various predisposing circumstances, produce a sudden change in the position of the organ which may be in a rotatory direction, and so start the twisting process.

Trauma, violent exercise, and sudden changes in body position may be considered as exciting causes. A blow to the abdomen may initiate the process directly or a resulting hematoma within the omentum may cause a sudden alteration in position and beginning twist. Coughing, straining, vomiting, etc., according to some authors may be responsible for the torsion. Prutz reports that changes in position occurring during active exercise, such as, running, jumping and acrobatics are exciting causes.

Increased Omental Movements: In D'Errico's opinion this is the causative factor in a number of his cases. There is some controversy as regards the mobility of the omentum. Some investigators state that its motion is derived from intrinsic factors; that it has "intelligent chemotaxis." Others claim that its motion is due to extrinsic influences. Of the latter group, Smyth, Morris and others find that the movements of the omentum are due to the action of the diaphragm, abdominal muscles, peristalsis of the gastro-intestinal tract and the state of distention or relaxation of the digestive organs. Normally, the omentum moves freely over the serous surfaces in the peritoneal cavity under these circumstances. Morestin states that increased peristalsis induced by enemata or catharsis may so affect omental movements as to cause twisting. Florey and Carlton did experimental work which further bears out the theory of the extrinsic nature of omental activity.

Thrombosis and Embolism of Omental Vessels: If thrombosis or embolism occurs in an omental vessel, the infarcted area serves to initiate the twisting because of the added weight due to the vascular block in the affected portion.

In concluding this discussion on etiology, it is our impression that while these exciting causes play a large part in the production of omental torsion, nevertheless, the predisposing factors mentioned are extremely important. Of these, we wish to stress the presence of anatomical anomolies.

INCIDENCE

Males have been reported as being more frequently affected by omental torsion than females. D'Errico, in thirty one cases, found only nine females, and the others in males. In a series of cases that we have

reviewed, we did not find this marked disproportion in sexes, nor did we find a greater percentage of males involved. In this series of twenty-seven cases there were fifteen females and twelve males. The condition may occur at any age, the greatest number of cases, however, falling between the ages of 30 and 50. The youngest case on record is a 5 year old child (see case report 2); the oldest is a man of 70.

PATHOGENESIS

With the process initiated by one of the factors discussed under etiology, it may terminate in one of three ways. First, with the cessation of the exciting causative factor the omentum may resume its normal position and function with no resulting pathological alteration. Second, the twisting may be of such mild degree that only slight changes occur in the vessels and omentum, which can be adequately compensated by the rest of the organ. Evidence of previous partial torsions have been found in a number of cases in which the patients gave vague histories of repeated abdominal complaints, such as pain, cramps, nausea, etc., extending over a period of months or years. At subsequent operation undertaken for acute severe torsion, the omentum was found to contain scar tissue, and many fibrous bands. These changes were undoubtedly due to the previous mild torsions which left the omentum in a state susceptible to acute torsion because of its irregularities, increased weight and malposition. Moynihan states, "Rotation in the omentum or other organs, as the ovary with a cyst, does not occur suddenly, the acute change is not the twisting of the pedicle, but the thrombosis of vessels which have long been twisted." This implies that frequently the acute process is the end result of a mild, chronic, recurrent torsion. Third, the twist may progress to complete infarction and gangrene due to the progressive vascular change. Farr and Bachmann claim that the initial twist may obstruct the lymphatics, cause edema which increases the weight of the affected portion, and acting like a plumet at the end of a string keep the process up until further circulatory obstruction causes gangrene.

SYMPTOMS AND SIGNS

The most constant feature in the symptomatology of these cases is the complaint of abdominal pain. This pain is usually severe and most frequently localized in the right lower quadrant. It may be generalized, or localized in any part of the abdomen. It may last from a few hours to as long as five days. In this series of cases, the average duration of pain was two to three days. The onset is, as a rule, sudden. The pain tends to be constant, non-radiating, and non-peristaltic in the greater majority of cases. Chart 1 indicates the various sites of the maximum intensity of pain.

D'Errico found vomiting present in about fifty per cent of the cases either before or after the onset of the pain. We found vomiting mentioned in the history in about twenty five per cent of the cases. Nausea is less frequent, being present in four of the twenty seven cases, an incidence of about 15 per cent. Neither constipation nor diarrhea is of frequent occurrence. In one of our cases, dysuria was a prominent feature due to the proximity of the twisted omental mass to the bladder. These patients, as a rule, do not appear severely ill. The temperature is usually elevated and there is a corresponding increase in the pulse rate.

Tenderness is always present, being most often elicited in the right lower quadrant (see Chart 1). It may also be found in the right upper quadrant or on the left side, depending upon the location of the pathology. A palpable mass has been reported in as high as forty per cent of the cases. In the case histories that we reviewed, a palpable mass was mentioned in eight out of twenty seven cases. In the two new cases which we submit, a mass was felt in the right upper quadrant of the first case, and by rectal examination in the pelvis of the second. This coincided with the site of maximum tenderness in each case. Various

degrees of rigidity may be present. This depends upon the degree of peritoneal irritation secondary to the pathological process affecting the omentum. We found it present in about 55 per cent of the cases studied. The greatest amount of spasm is most frequently located in the right lower quadrant (see Chart 1). This occurrence is explained, as is the pain, tenderness and location of the mass by the fact that most of the cases were of right sided omental torsion.

CHART I

Localization of Pain	Cases	Site of Tenderness	Cases	Site of Rigidity	Cases
Right lower quadrant	8	Right lower quadrant	18	Right lower quadrant	18
Epigastric	5	Right upper quadrant	6	Right upper quadrant	5
Generalized abdominal	4	Left lower quadrant	1		
Right side of abdomen	4	Lower abdomen	1		
Lower abdomen	3				
Right upper quadrant	1				
Left side of abdomen	1				
Umbilical	1				

The blood count may reveal varying degrees of leucocytosis ranging as high as 17,000. This is dependent upon the extent of the pathological change and requires no special comment.

PATHOLOGY

In the majority of cases the right half of the omentum is involved, although in some the whole organ is affected. The torsion occurs usually in a clock-wise direction, with the number of twists varying from one-half to twelve or fourteen complete turns. The twisted omentum may form a rope-like structure or a rounded mass which grows larger with each turn. There are three stages in the process of torsion. First, congestion with engorgement of the veins and capillaries; second, thrombosis of the vessels with infarction of the involved portion of the omentum; third, gangrene and necrosis. Microscopic sections of twisted omenta show congestion, stasis, and thrombosis in the blood vessels with necrosis of the fat material and hemorrhage.

Upon opening the abdomen in a case of torsion of the omentum, there is usually found an increased amount of peritoneal fluid which may be serous, turbid or sanguinous. At the site of the pathology a mass is found which appears bluish-black in color and adherent by fine, filmy adhesions to the parietal peritoneum or the abdominal viscera. These are usually separated very easily, and the point of demarcation of the gangrene can be visualized. In cases of bifid omenta, where one limb only is involved, the other free portion of the omentum is found wrapped around the diseased part, in its customary attempt at limitation of an intra-abdominal pathological process.

DIAGNOSIS

The correct diagnosis has rarely, if ever, been made preoperatively. The most common preoperative diagnosis is appendicitis. This was the case in twenty out of the twenty seven cases reviewed. When we consider that the pain, tenderness and rigidity are so frequently located in the right lower quadrant, we can easily realize why appendicitis is the most frequent diagnosis. Gall-bladder disease ranks second as a diagnosis (see Chart II).

A differential diagnosis between primary omental torsion and appendicitis is difficult to make. D'Errico, Jeffries, Huff and Farr and Bachmann all stress the fact that gastric symptoms such as nausea and vomiting are rarely present in primary omental torsion. One very important feature, as pointed out by most authors, is the

rapid development of a mass in torsion of the omentum. This may appear suddenly and certainly far more quickly than one would expect in an acute appendicitis. The majority of these patients are somewhat obese and robust, and careful questioning may elicit a history of some sudden exertion immediately or soon followed by abdominal pain.

In studying the case histories, we find that the following factors are somewhat constant, and either singly or in any combination should lead one to suspect the presence of this condition: history of some marked exertion; sudden onset of constant, non-radiating, non-peristaltic abdominal pain; slight, if any, nausea and vomiting; rapid appearance of an abdominal mass.

Chart II

PREOPERATIVE DIAGNOSES

Acute appendicitis	14
Acute cholecystitis	4
Galibladder or appendix	2
Chronic appendicitis	2
Appendiceal abscess	2
Diverticulitis with abscess	1
Ovarian cyst	1
Surgical abdomen	1
Total cases	27

TREATMENT

The treatment is strictly surgical, with a resection of the involved portion of omentum. The mortality is extremely low. The few deaths that have been reported have been due to pneumonia, emboli, and other accidents that make up the general operative mortality.

CASE REPORTS

Case I. (Fig. 1.) H. F., male, aged 40, was admitted to Beth Israel Hospital on August 19, 1935. Three days before admission the patient was suddenly seized with severe abdominal pain, mainly in the right lower quadrant. The pain was gnawing in character with occasional periods of exacerbation lasting from one-half to one hour. He took a physic on the following day with relief of pain. On the day before admission he had a recurrence of the pain with sour eructations. There was never any diarrhea, nausea or vomiting.

Examination revealed a middle aged, obese male complaining of abdominal pain. Temperature 103.4, pulse 76 and respirations 30. General examination was negative. Abdominal inspection revealed a distended, protuberant abdomen with tenderness, rigidity and peritoneal release in the right lower quadrant, and release in the left lower quadrant. There was the sensation of a mass in the right upper quadrant, where some tenderness was also elicited.

Urine examination was negative; blood count revealed 9,950 white blood cells with 84 per cent polys and 2 per cent staffs.

Diagnosis of acute appendicitis made and operation performed. A lower right rectus muscle splitting incision was made. On opening the peritoneal cavity there escaped a moderate amount of serosanguinous fluid. The omentum which presented in the wound was deeply congested and gangrenous in areas. Palpation of the right upper quadrant revealed a large mass which could be delivered into the wound only after enlarging the incision. The mass proved to be the result of a torsion of the greater omentum involving the distal portion. It was twisted eleven and one-half times. The omentum was of the bifid variety, with torsion of the right limb, surrounded by the free left portion. No inflammatory or other pathological process was found in the abdomen. The diseased omentum was resected, and the abdomen closed in layers.

The pathological diagnosis was hemorrhagic infarction of omentum. The specimen consisted of a large apron-like mass of omental tissue, the central portion of which is foreshortened and shows fibrous streaks, suggesting that the torsion was probably chronic in nature.

The postoperative course was uneventful until the fourteenth day, when the temperature rose to 101 and continued to vary between 100 and 101 until the twenty fifth day. No cause for the fever could be determined. The patient was discharged cured on the twenty eighth day, and has remained well up to the present.

Case II. Report of youngest case published.

E. C., female, aged 5, was admitted to Beth Israel Hospital on July 15, 1936 complaining of abdominal pain for twenty six hours. After repeated questioning, the following interesting facts were elicited concerning her earlier years. As an infant she frequently had attacks of apparent abdominal pain diagnosed at that time as "colic." Since then, she has had occa-

sional attacks of abdominal cramps associated with nausea. The past history is otherwise negative. Twenty-six hours prior to admission the patient was suddenly seized with pain in the mid abdomen, and was given mineral oil. The pain disappeared, and the child resumed playing in the street. The pain recurred in twelve hours, and an enema was given with no relief. The pain persisted up to time of admission and was accompanied by vomiting. The pain was referred to the suprapubic region, and was associated with dysuria.

Examination showed an acutely ill appearing child, lying in bed with thighs flexed. The general examination was negative. The abdomen was slightly distended. Palpation revealed spasm in the lower abdomen with tenderness and rebound tenderness especially in the suprapubic region and to the right. Rectal palpation showed the presence of a boggy mass in the right rectal fornix.

Diagnosis of acute appendicitis, probably with abscess adjacent to or involving the bladder was made, and the patient prepared for the operating room. The temperature at this time was 101.4, respirations 32 and pulse 130. Urine was negative except for frequent white blood cells. White and differential blood count revealed a leucocytosis of 14,400 with 83 per cent polys and 4 staffs.

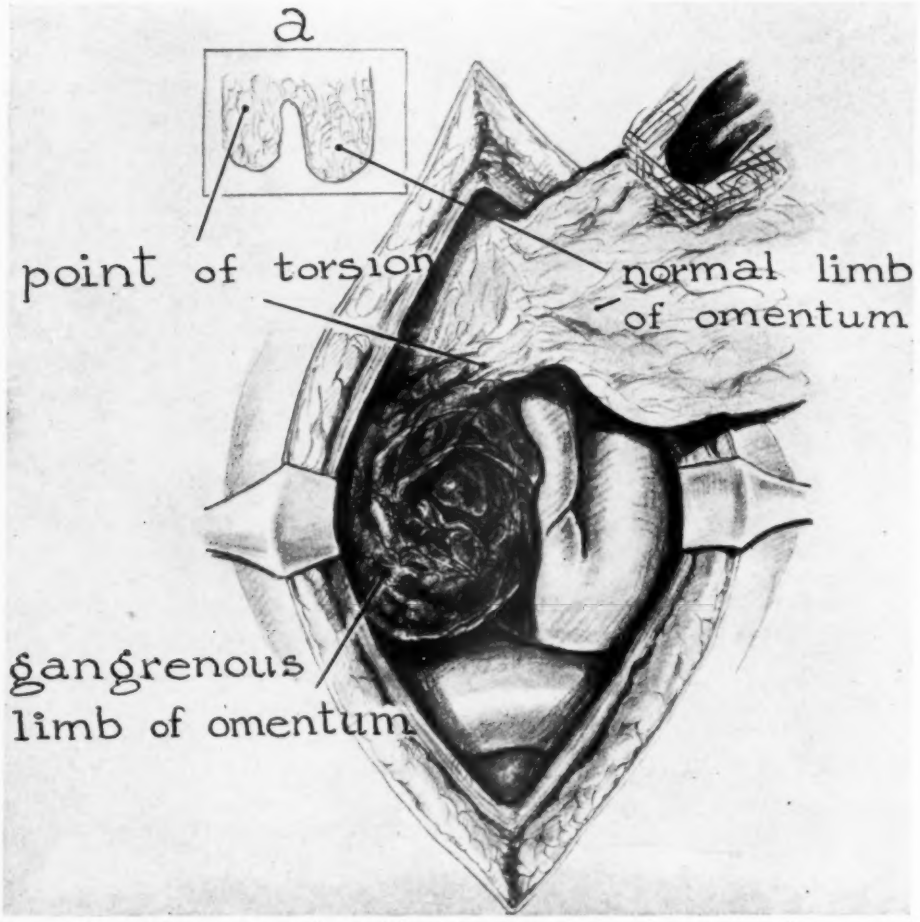


FIG. 1. Case 1. Gangrenous, twisted right limb of omentum as seen at operation, exposed by elevation of the normal left limb of omentum. a; diagrammatic representation of bifid omentum showing point of torsion.

At operation a right lower rectus incision was made with lateral retraction of the muscle. A small amount of serous fluid was found in the peritoneal cavity. In the right side of the pelvis behind the broad ligament was a gangrenous, inflamed mass with filmy adhesions to the pelvic wall. This was at first thought to be an appendiceal abscess, but on further examination the appendix was found to be retrocecal and bound down by adhesions, and not a part of the mass. The mass was then delivered into the wound and was found to be the distal por-

tion of the omentum attached by a thin, attenuated string of omentum to the main portion, and twisted five and one-half times upon itself. No inflammatory or other pathological condition was found in the abdomen to account for the twisting. The gangrenous omentum was excised, and an appendectomy performed. The pathological report was infarction of omentum with hemorrhage. The patient ran an uneventful postoperative course, and was discharged cured on the thirteenth day. She has remained well to date.

SUMMARY

Review of the literature reveals sixty cases of primary omental torsion reported to date.

This report is based on a study of twenty five of the above cases plus two of our own.

We submit detailed reports of two additional cases, one the youngest on record.

Anatomical variations are of fundamental importance as a predisposing cause.

A definite symptom-complex is presented which, it is felt, will aid in the correct diagnosis.

REFERENCES

1. Smyth, M. J. Abdominal Torsion of the Omentum. *Lancet*, 2:572, Sept. 13, 1930.
2. Morris, J. H. Torsion of the Omentum. *Arch. Surg.*, 1: 40, 1932.
3. Jeffries, J. W. Torsion of Great Omentum. *Ann. Surg.*, 93: 761, 1931.
4. D'Errico, W. Primary Torsion of the Great Omentum. *New Eng. M. J.*, 203: 1181, 1930.
5. Allen, L. Primary Torsion of Omentum. *New Eng. M. J.*, 209: 235, 1933.
6. Rochelle, F. O. Torsion of Omentum Simulating Appendicitis. *American J. Surg.*, 20: 97, 1933.
7. Huff, W. B. Torsion of Omentum with Signs and Symptoms of Acute Appendicitis. *Virg. M. Monthly*, 57: 583, 1930.
8. Wallace, R. H. and Miller, R. H. Primary Omental Torsion. *New Eng. M. J.*, 208: 831, 1933.
9. Benton, J. L. Idiopathic Torsion. *Wis. M. J.*, 33: 204, 1934.
10. Cromie, D. Idiopathic Torsion. *Brit. M. J.*, 1: 106, 1935.
11. Farr, C. E. and Bachmann, R. F. Torsion of Omentum. *Ann. Surg.*, 97: 766, 1933.
12. Betz, H. Etiology of Omental Torsion. *Zentralblat. f. chir.*, 60: 1409, 1933.
13. Robertson, J. H. Torsion of Omentum. *Tr. Royal Med. Chir. Soc.*, p. 72, Glasgow, 1933.
14. Robb, D. Abdominal Torsion of Omentum. *Lancet*, 2: 1347, 1930.
15. Weeder, S. D. Torsion of Omentum. *S. Clin. N. A.*, 11: 1443, 1931.
16. Speese, A. Omental Cysts. *Internat. Clin.*, 30: 246, 1920.
17. Laurence, S. Cyst of Omentum. *South. M. J.*, 18: 423, 1925.
18. Eitel, B. Omental Torsion. *Med. Rec.*, 55: 715, 1899.
19. Niemerer, W. Torsion of Omentum. *Canad. M. J.*, 32: 175, 1935.
20. Lewis, D. Practice of Surgery. 7: 8, Chap. 8.
21. Hertzler, B. The Peritoneum. St. Louis, C. V. Mosby Co., 2: 750, 1919.
22. Arey, L. B. Developmental Anatomy. N. Y., W. B. Saunders Co., p. 124, 1926.
23. Cunningham, H. Manual of Anatomy. London, Oxford Univ. Press, 2: 266, 1930.

PYODERMIA COMPLICATING ULCERATIVE COLITIS

MAX P. COWETT, M.D.

NEW YORK CITY

THE term pyodermia denotes a purulent infection of the skin due to a pyogenic organism, usually staphylococci. Of the skin lesions complicating ulcerative colitis one may encounter furunculosis or erythema nodosum. The occurrence of a real pyodermia is relatively rare.

In 1929, Glaubersohn[1] described a definite clinical picture in which a constant triad of symptoms was present—ulceration of the bowels, ulcers of the skin and a hypochromic anemia. Most of the patients were young girls. Brunsting, Goeckeman and O'Leary[2] first reported five cases in 1930 with a similar picture, naming it pyodermia gangrenosum. Lane and Malone[3] in 1933 also described this condition. In 1936, Jankelson and Massel[4] reported five cases and Cohen[5] gives an exhaustive report of a case. There have been other cases reported but the condition is rare.

There have been various causes ascribed to the pyodermia associated with ulcerative colitis. It is believed to be either a deficiency disorder, due to avitaminosis, or there may be a metastatic origin of the skin infection. Another thought is that the skin condition may be caused by the synergistic action of streptococci and staphylococci. However, the writer does not accept Schwartzman or Arthus phenomenon as a causative factor.

REPORT OF CASE

S. L., female, aged 27 years, married, was first seen in December, 1935, because of bloody diarrhea, four to eight movements a day, of four months' duration.

Previous History: One child, living and well, at age of 4. Negative previous history except for sinusitis. At the age of 8 she was said to have had rheumatism of lower limbs for one week but no recurrence.

Gastro-intestinal History: As a child, vomited frequently, which was attributed to a post nasal drip. She has been constipated all her life until onset of present illness. The patient has never been outside of New York State in her life.

Present Illness: In August, 1935, she began to have four to five stools a day, consisting of very small movements. Also after two or three weeks, noticed slight blood and mucus. She was treated with a roughage diet and as a result developed a real diarrhea (ten to fifteen bloody stools a day). She began to lose weight and became weak. In December, 1935, she was referred for rectal and proctoscopic examination which revealed a small anterior skin tab and small superficial fissure. Proctoscopy showed a slight hemorrhagic proctitis but no gross ulceration was visualized. Scope passed 16 cm. with considerable discomfort. Smears were taken and one amebic cyst was seen. She was sent back to the referring phsyician who treated her by Emetine, Chiniofin, high caloric and low roughage diet. The diarrhea subsided and she felt greatly improved. She was not seen again until March, 1936, when there was a recurrence. Proctoscopy at this time showed the typical hyperemic stage of ulcerative colitis but no evidence of amoebic ulceration. Roentgenograms were negative and showed a normal sigmoid and colon. She was treated with calcium gluconate, gr. 11 three times daily, two hours after meals with Kaolin and liver and iron and Viosterol, also with rectal irrigations of $KMnO_4$, gr. 11 to two quarts of water. The patient improved, bowel movements decreased to one or two a day and she gained seven pounds in weight.

When last seen on July 12th, 1936, she was inclined to constipation and had cramps for the last two days with slight bloody mucus. Red blood cell count was 4,170,000 and hemoglobin was 81 per cent. Proctoscopy again showed the same picture.

The patient was admitted to Mount Sinai Hospital on August 24th, 1936, on the service of Dr. Burril Crohn.

Physical examination revealed a thin, sallow female, who appeared to be chronically ill. Temperature was 99°, respirations 18, pulse

rate 90, regular. Blood pressure was 108/70; weight 105 pounds. Physical examination was essentially negative except for abdomen which was soft and somewhat distended. Bilateral lower quadrant pain on deep pressure and here, on both sides, distended bowel could be felt.

Rectal examination revealed external skin tab. Sigmoidoscopy showed an appearance typical of ulcerative colitis.

LABORATORY EXAMINATIONS:

BLOOD COUNTS

	8/25	9/4	11/18	11/21	11/24	12/4	12/17	12/30
Erythrocytes	3,680,000	.....	2,280,000	2,700,000	....	2,840,000	3,170,000	3,440,000
Hemoglobin	53%	50%	32%	40%	40%	54%	60%	60%
Leucocytes	9,500	20,900	12,800					
Polys	52%	42%	65%					
Lymphocytes	30%	40%	23%					
Mononuclears	5%	9%	2%					
Eosinophiles	13%	9%	2%					
Myelocytes & neutrophiles	.....	.....	8%					
Polychromatophilia	.....	.....	some					
Achromia	.....	.....	moderate					
Anisocytosis	.....	.....	moderate					
Poikilocytosis	.....	.....	moderate					

Wassermann and Kahn tests: Negative.

Blood cultures November 18—no growth after 96 hours. November 25—no growth after 72 hours.

Blood Agglutinations: August 26th—Negative for five strains of dysentery.

Stools: Daily stool examinations were negative for amoeba and parasites. Only findings were pus, 2 plus and red blood cells 4 plus.

Barium enema: August 26th,—spasm of sigmoid and descending colon. No irregularities of colon, but after evacuation the mucosal pattern of the descending colon and distal transverse colon is distorted, suggesting an ulcerative colitis.

Normal horse serum and intracutaneous test: August 28th—negative.

Urinalyses: essentially negative.

CLINICAL COURSE AND TREATMENT

On August 28th, 29th, and 30th, given 5, 10 and 10 cc. of antidysentery serum. There was no reaction except that number of bowel movements increased. On August, 31st, twenty million typhoid given intravenously. Temperature rose to 104.4. On September 4th, urticaria and itching developed over body, ascribed to serum. On September 9th, ten million typhoid vaccine intravenously. Temperature rose to 104.8. On September 14th, 500 cc. blood tranfusion was given. During her hospitalization, she received a series of emetine (six doses, gr. 1), carbasone tablets, deodorized tincture of opium and reduced Iron, gr. x, three times daily.

Final note: September 23rd, 1936. "A 27 year old housewife, previously well, was admitted with one year history of bloody, mucoid diarrhea, occurring in repeated attacks of variable duration. She was given gradually increasing doses of dysentery serum intravenously followed by a course of typhoid vaccine. Subsequently, transfused on one occasion. Despite slight improvement, she was discharged at her own request to complete course of treatment at home."

Subsequent history: The patient continued to have abdominal cramps and five to ten bloody mucoid stools with gradually increasing weakness. On November 7th, 1936, she was given another transfusion of about 150 c.c. Three days later, she developed a pustular eruption in the right antecubital fossa.

I saw her again on November 14th, 1936. She looked acutely ill with temperature of 101.4° and pustules were present (three or four over right leg and two over the anterior surface of the right forearm).

The right knee joint was swollen and tender.

She was admitted to New York Polyclinic Hospital on November 17th, 1936, acutely ill and toxic. The temperature was 102°, pulse rate 120, respirations 22, blood pressure 90/58 and there was a pustular eruption over both lower extremities, on both arms and a smaller one on left cheek. The following day her temperature rose to 104.6° and for about three weeks she ran a septic temperature ranging from 102° to 106°. Her general condition was poor, pulse of extremely poor quality, rapid and irregular. Both knee joints were tender and swollen and tenderness over the right sternoclavicular joint was also present. She was given four blood transfusions, 450 cc., 350 cc., 350 cc., 300 cc. on November 19th, 21st, 27th and December 5th, with improvement in her general condition after each transfusion. Because of the finding of the hemolytic streptococcus in the pustules, she was given an intramuscular injection of 5 cc. prontosil daily from November 21st to November 26th.

At first, boric acid warm compresses were applied to the ulcerated areas but because the patient complained of severe pain, lead and opium wet dressings were applied. The weight of the dressings could not be tolerated and they had to be removed. Balsam Peru, carbolated vaseline were tried as well. She was also immersed in warm baths in order to remove the slough.

FIG. 1. Left leg, showing diffuse ulceration with areas of granulation.

On November 28th, allantoin spray was used on the ulcerations of the skin, and it seemed to help in removing the sloughing areas.

On December 3rd, azochloramide in oil was started and this also seemed to help in the healing process of the skin ulcers.

When the patient was first admitted to the Polyclinic Hospital she had from six to twelve bowel movements, always associated with abdominal cramps and tenesmus. It was noticed that as she became toxic, her

bowel movements decreased in number and became more formed. Her diarrhea practically subsided by December 10th, 1936.

rounded by an inflammatory areola and rapidly became confluent. Within a few days, the center of the pustule softened

Fig. 2. Right leg, showing serpiginous type of ulceration.

No attempt had been made to proctoscope the patient because of her poor general condition, but on December 20th and December 29th, no ulcerations were visualized as high as 18 cm. The passing of the proctoscope was attended with a moderate amount of spasm, but the mucous membrane looked healthy and no evidence could be found of any ulcerative condition of the colon.

She was discharged on December 30th, 1936, markedly improved. The skin lesions have not entirely healed but are rapidly filling in. She complains of moderate abdominal cramps but has had no diarrhea for two weeks. Her weight on leaving the hospital was 70 pounds, a loss of 35 pounds.

The skin lesions presented an unusual appearance. They involved both legs and arms and a small one on the left cheek, none on the thorax or abdomen. Pustules when first noted had a bluish color, were surand the covering skin became blue and broke down. The lesion either underwent involution or extended to coalesce with those adjoining to form a large ulcerative process. There was extensive undermining and necrosis of the subcutaneous tissue. The lesions spread rapidly, often at the rate of 2–4 cm. within twenty-four hours. The base was moist and covered with a mucopurulent exudate, which exuded from the necrotic area. The lesions at six or seven of the sites extended down to the muscle, laying it bare.

Healing took place by epithelial outgrowths with a resulting thin, atrophic scar which had a brownish-blue pigmentation and after healing, maintained their serpiginous outline.

Smears and cultures obtained from secretion of ulcers showed the following organisms:

Staphylococcus albus,

Streptococcus hemolyticus,
Cocci in clumps,
Gram negative bacilli.

On account of the rapidity of spread of the ulcerative lesions, I had to denude and excise considerable amount of the undermined edges on many of these ulcers.

COMMENT

The purpose of reporting this case is that the author did not associate the pyogenic skin infection with the colitis, but ascribed it as being due to a local skin infection, the pyodermia developing at the site of the last transfusion, which had been given three days previously.

While one amebic cyst was found on proctoscopic examination, it is the author's opinion that this patient did not have amebic colitis. She was in the diarrheal stage and amebic cysts are rarely if ever found in this stage—only when the patient is constipated.

Another point to be stressed in this case is that this patient seems to have developed an autogeneous vaccine within her own body which relieved her condition greatly. She was extremely septic for about three weeks with the described ulcerative condition of her extremities. Following this, her diarrhea decreased and proctoscopy showed no ulceration. She has continued to improve and her skin lesions have all healed, although there is considerable scarring. Up to the present (March 17th, 1937) there has been no recurrence. The patient has one or two normal bowel movements a day. The blood which is present only rarely, is probably due to a small fissure. Her weight on leaving the hospital on December 30th, 1936, was 70 pounds and on March 17th, 1937, she weighed 88½ pounds.

URETHRO-PROSTATIC FISTULA

REPORT OF TWO CASES

J. W. VISHER, M.D.

EVANSVILLE, INDIANA

SINCE a review of the standard text books on Urology reveal scant mention of the subject of this paper, one is forced to the conclusion that these cases are rare.

FIG. 1. Radiogram showing large calculus in prostatic region.

FIG. 2. Radiogram showing bladder filled with campiodal emulsion, and urethral catheter beside stone in prostatic urethra.

My first case is a farmer, age 24, who sat down forcibly on a corn stalk, which penetrated into his rectum and entered his prostatic urethra. Urinary retention persisted for eighteen hours, and upon straining, the urine escaped freely into the rectum. Retention again supervened, and urethral catheterization by Dr. J. V. Ferrell was unsuccessful. I saw him about thirty-six hours after the accident, at the Ferrell Hospital, Eldorado, Illinois. At that time, the bladder was moderately distended, but there was no evidence of urinary extravasation.

I at once performed a suprapubic cystotomy and carefully explored the bladder and found it normal. An attempt at retrograde catheterization was unsuccessful, as also was anterograde catheterization with an ordinary catheter. A soft rubber coude catheter was then passed without much difficulty, and its distal end was brought out through the suprapubic wound and fastened securely to its proximal end, making a complete circle. A Pezzar catheter was then introduced into the bladder through the suprapubic opening, and the wound was closed loosely around both catheters. The post-operative course was uneventful except that intestinal gas escaped through the suprapubic tube at intervals for about fourteen days after the operation. The indwelling urethral catheter was removed in twelve days and the suprapubic tube in twenty-one days. The suprapubic wound was completely healed forty days after the operation, urination was normal, and there was no drainage of urine into the rectum. An important point in the after care was periodic passage of sounds to prevent stricture formation.

My second patient is a man 60 years of age, whose health was good until about fifteen years ago when he began to have painful and frequent urination. About six years ago he first noticed, during urination, that some urine passed into

his rectum. These symptoms gradually became worse until at the time of his admission to St. Mary's Hospital June 2, 1936, he had a great deal of pain and localized soreness in the rectal region. Rectal examination showed the prostate to be hard, but not enlarged, and there was a depressed area near the anal opening. Attempts to pass a catheter into the bladder were unsuccessful. A urogram showed no evidence of renal or uretheral calculus. Apparently there was a stone in the prostatic urethra, about 1 inch long and a half inch wide. (Fig. 1) A catheter was passed as far as possible into the urethra and the bladder, was filled with campiodal solution, and anteroposterior and lateral radiograms were taken. These showed definitely that the stone was in the prostatic urethra. (Figs. 2 and 3) The lateral also showed some of the solution in the rectum.

Fig. 3. Lateral radiogram (retouched) showing stone with catheter in front of it. The bladder filled with campiodal emulsion is faintly outlined above the head of the femur, behind which some of the emulsion is seen in the rectum.

Under gas-ether anesthesia, I performed a suprapubic cystotomy and explored the bladder. There were no stones within the bladder, but by introducing the finger into the prostatic urethra the stone was found. The prostatic urethra was then gently dilated by the finger and the stone was removed with sponge forceps. It left a large pocket within the prostate, formed by dilation of prostatic urethra. There was a large amount of scar issue, especially on the rectal side. A Pezzar catheter was introduced into the bladder and the wound was closed loosely around it. The patient made a smooth convalescence from the operation until the sixteenth day, at which time, under evipal anesthesia, I passed a sound into the bladder in order to dilate the prostatic urethra. This was followed by chills and elevation of temperature for several days. However, he recovered sufficiently to go home four days later.

He returned September 19, 1936, reporting that he had improved satisfactorily for the first month after going home, but after that, his urinary difficulties returned and the suprapubic wound broke open and drained urine. Rectal leakage was infrequent and very small in amount. Under evipal anesthesia I again dilated the urethra to size 24, and thoroughly treated the wound with silver nitrate. He remained in the hospital only a week, and at the time of leaving, the suprapubic wound had closed and urination was free and painless.

A report was received from his family physician, Dr. E. W. Cummins of Harrisburg, Illinois, on November 7, 1936, in which he states that the patient is very much better in every way. He still has a small amount of urinary drainage into the rectum occasionally, whereas, before the operation, it occurred at every urination.

COMMENT

These two cases illustrate what can be accomplished by conservative treatment. Prolonged suprapubic drainage allowed healing of the fistula in the first case and almost complete closure in the second. Improvement should be permanent, provided they return at regular intervals for urethral dilation.

TRAUMATIC DIAPHRAGMATIC HERNIA

REPORT OF A CASE

WILLIAM H. MAST, M.D., F.A.C.S. AND J. P. MCDONOUGH, M.D.

GUNNISON, COLORADO

PENETRATING injuries of the lower thorax or upper abdomen are frequently associated with trauma to the diaphragm. Intestinal obstruction may occur through the new-formed hiatus or the diaphragmatic injury may be slight and remain unrecognized. Later, due to various factors such as childbirth, straining or strenuous exercise, a hernia may follow resulting in acute intestinal obstruction.

The importance of recognizing diaphragmatic injuries, and the unusual occurrence of such herniae subsequent to a previous trauma are well illustrated by the following case report:

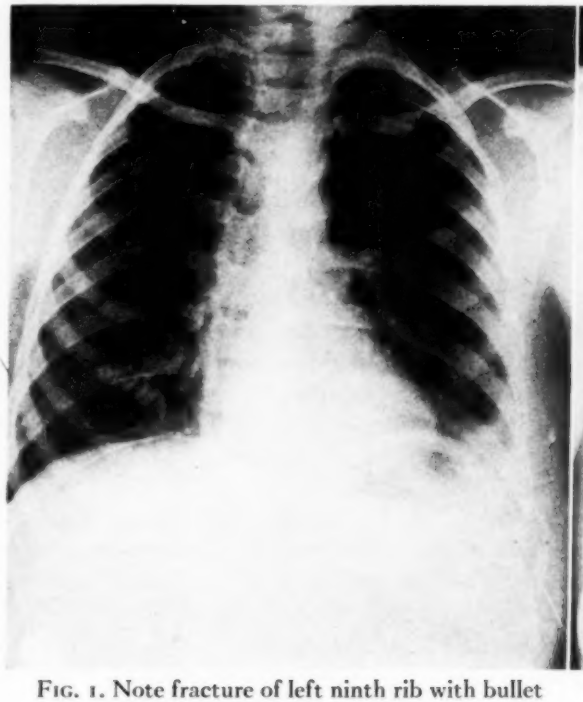


FIG. 1. Note fracture of left ninth rib with bullet fragments in soft tissue.

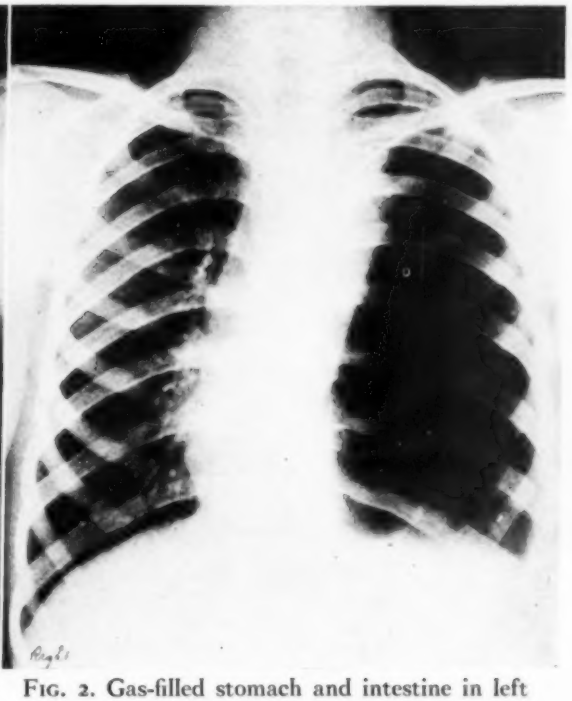


FIG. 2. Gas-filled stomach and intestine in left thoracic cavity. Displacement of the heart and mediastinal structures to the right.

CASE REPORT

White male, age 13. Admitted to the hospital August 10, 1936, two hours after an accidental gunshot wound of the upper abdomen and lower thorax. The past history and family history were entirely irrelevant.

A .22 long bullet had entered the abdominal wall 2 cm. below the ensiform cartilage. It passed in a transverse and superior direction, emerging at the level of the eighth rib in the left mid-axillary line. Roentgen examination taken at that time (Fig. 1) revealed a comminuted fracture of the left ninth rib, three inches from its anterior extremity.[1] There were opaque particles (fragments of the bullet) in the soft tissues external to the fracture and leading through the soft tissues of its internal side. The course of the bullet could not be determined on the flat plate. There was no gross pathology visualized in the lung fields.

The patient was not in shock and showed no sign of visceral injury. The wound was cleansed

[1] *X*-ray interpretations by Dr. G. A. Unfug, Pueblo, Colorado.

and protective serum administered. He remained in the hospital for two weeks, during which time the temperature varied from 99°F.

FIG. 3. Necropsy examination showing: (A) stomach; (B) gangrenous portion of the ileum which has been displaced from the thoracic cavity in order to demonstrate the stomach; (C) lung, and (D) index finger in diaphragmatic hiatus.

to 102°F., gradually becoming normal. There was moderate drainage from the wound which was completely healed at the end of three weeks. During the first five days the patient complained of severe pain in the left shoulder. Codein was required several times for relief of this pain. No significance was attached to this symptom, and a check *x*-ray of the chest upon discharge showed no change from the former examination.

The patient was examined at the office on Feb. 11, 1936, six months following the original injury. He was extremely well and had gained six pounds. His appetite was good and the gastrointestinal and respiratory systems were normal.

April 8, 1936, eight months after the original injury, the patient was admitted to the hospital in a moribund condition. Four days prior, April 4, while skiing, he rolled down the mountain side. That evening he noticed pain in the epigastrium, aggravated by lying down. The pain was referred to the left shoulder. It was sharp but relieved by heat.

April 5, the pain was lessened, but he vomited several times. The following day, April 6, he regurgitated small amounts of odorless, brownish fluid. He had only slight pain this day.

On April 7, he was much weaker as he was now unable to retain any food. That evening, the pain in the epigastrium and left shoulder became more severe and vomiting occurred every hour. No physician was called and on April 8, he was taken from his home to the hospital, a distance of seventy miles.

Physical examination revealed a white boy of stated age, 13. He was lying in bed and extremely ill. There was moderate cyanosis of the lips and finger tips. The respirations were 46, temperature 102.6°F. and pulse 136. Orthopnea was marked. The patient responded readily to questions and he was well oriented.

Examination was directed particularly to the thorax and abdomen which revealed the following significant findings:

Cardiovascular system: Pulse 130 beats per minute, regular but of poor quality. Blood pressure 90/42. P.M.I. in the fourth interspace 2 cm. to the right of the midclavicular line. The right border of cardiac dulness was percussed 2 cm. to the right of the right sternal margin.

Thorax: A scar was present in the left midaxillary line representing the former gunshot wound. There was diminished excursion of the left thorax with loss of the normal intercostal depressions and fixation of the costal margin of the left side. Tympany was noted anteriorly over the lower thorax and dulness posteriorly (patient semi-recumbent).

Lungs: The breath sounds were diminished over the lower portion of the left thorax with some increase of the respiratory murmur above the third rib anteriorly and posteriorly. A marked succussion splash was to be heard over the lower left thorax.

Abdomen: There was generalized involuntary rigidity and tenderness present, particularly noted in the right upper quadrant. A small scar just below the ensiform cartilage represented the former gunshot wound.

The leucocytes numbered 23,800, with 90 per cent polymorphonuclear cells, 65 per cent of which were non-segmented. Urinalysis: turbid, amber, specific gravity 1.028, 2 plus albumen, no sugar present. Examination of the sediment revealed 1 plus pus cells.

Roentgen examination of the chest (Fig. 2) was as follows: There are irregular areas of decreased density occupying the major portion of the left chest, the largest area extends the length of the thoracic cavity and is about 2 inches in width. No lung markings are visualized in these areas. The heart and mediastinal structures are displaced to the right. The entire left lung is displaced upward and to the right. These findings are believed to be the result of herniation of the stomach or intestines, which are distended by gas, through the left hemidiaphragm. The right lung appears to be negative by *x*-ray.

The patient was placed under an oxygen tent and stimulation and intravenous glucose in 50 per cent solution administered. Attempt was made to relieve the gas in the herniated bowel by thoracentesis. Some gas was obtained, but the condition of the patient grew progressively worse and he expired eighteen hours following admission.

Permission for a partial necropsy was obtained and revealed the following pathology: The entire stomach and eighteen inches of the mid-portion of the ileum were present in the left thoracic cavity (Fig. 3). Herniation had occurred through the central portion of the diaphragm. The stomach was markedly distended with gas and fluid and was quite discolored. The intestinal loop was displaced from the thoracic cavity during the examination to demonstrate the stomach. This portion of the ileum was distended to four times normal size. The wall was friable and gangrenous. The left lung was atelectatic and normal crepitation was absent. About 150 cc. of straw-colored fluid was present in the left thoracic cavity.

The index finger of the right hand is inserted into the diaphragmatic hiatus. The opening admitted two fingers, and the stomach at the pyloric outlet and the small intestine were firmly incarcerated at this point. The margins of the ring were friable and composed of scar tissue. About 500 cc. of straw-colored fluid was present in the abdominal cavity. The remaining ileum below the obstruction showed some distention, but no other intra-abdominal pathology was noted.

Comment: The initial diaphragmatic injury which occurred at the time of the gunshot wound was entirely overlooked. The association of pain in the left shoulder as well as the course of the bullet might have suggested such a trauma. An exploratory operation after recovery, with repair of the injury would have seemed to be indicated.

CONCLUSIONS

1. A case of traumatic diaphragmatic hernia resulting in intestinal obstruction is presented. The hernia occurred eight months following the initial injury to the diaphragm.

2. The characteristic roentgen and physical signs of diaphragmatic hernia with incarceration of the stomach and intestines are noted in the case presented. Particular attention is directed to the association of shoulder pain with diaphragmatic trauma.

3. Penetrating injuries of the lower thorax or upper abdomen may be associated with diaphragmatic injury. Diaphragmatic hernia and intestinal obstruction may occur immediately or follow the original injury by various intervals of time. In the presence of suggestive signs and symptoms of diaphragmatic injury, an exploratory operation might more frequently be employed to prevent a disastrous outcome such as occurred in this case.

SEVERE CRUSHING FRACTURE OF VERTEBRAE WITH COMPLETE RECOVERY

A. E. GORDIN, M.D. F.A.C.S.

Attending Surgeon Jackson Infirmary, Mississippi Charity Hospital

JACKSON, MISSISSIPPI

CASE HISTORY

ADMITTED, March 22nd, 1930. A colored man, 28 years old, single, laborer, six feet in height, weight 165 pounds, with a negative past history.

Present history: While he was waiting in an empty room where certain fertilizers are stored, a car of acid material was dumped in the room, almost completely covering him. After getting him out he was found to be badly burned and complained of severe pain in back. For this reason he was brought to the hospital in the ambulance.

Symptoms and examination: On admission to the hospital an examination revealed that this patient was badly burned around the shoulders, upper part of back and arms. He was not in shock nor did he have any paralysis. However, he complained of severe pain in the lumbar region of back and complained of partial anesthesia in both legs. Examination was otherwise negative.

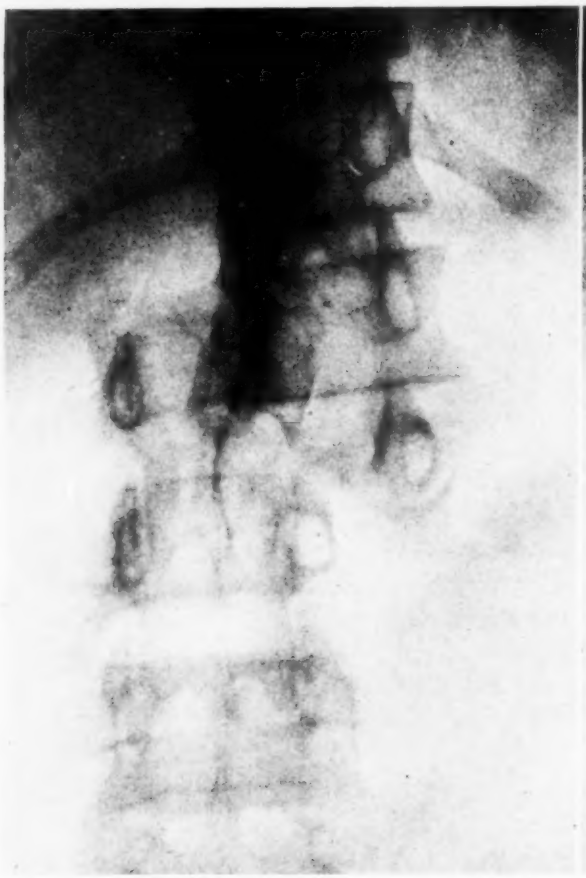


FIG. 1. Shows original fracture at time of admission to hospital.

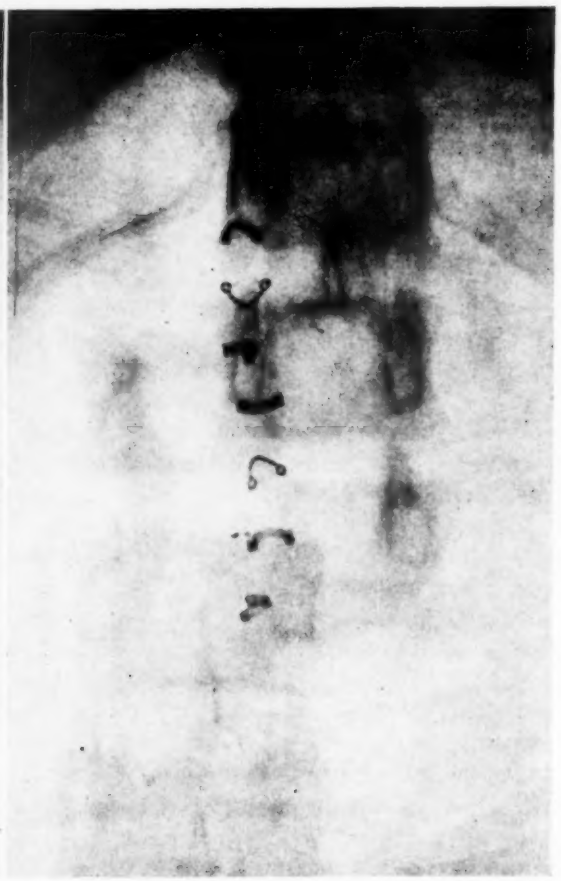


FIG. 2. Shows fracture after operation with posterior structure of second and third lumbar vertebra removed and cord replaced.

X-ray examination report as follows: A single radiograph and a stereoscopic radiograph of the lumbar spine show multiple fractures of the second and third lumbar vertebrae (See Fig. 1.)

Procedure: Because of the patient's severe burns it was impossible to reduce the fracture and also impossible to put on any form of brace or cast, so it was decided to do an open operation.

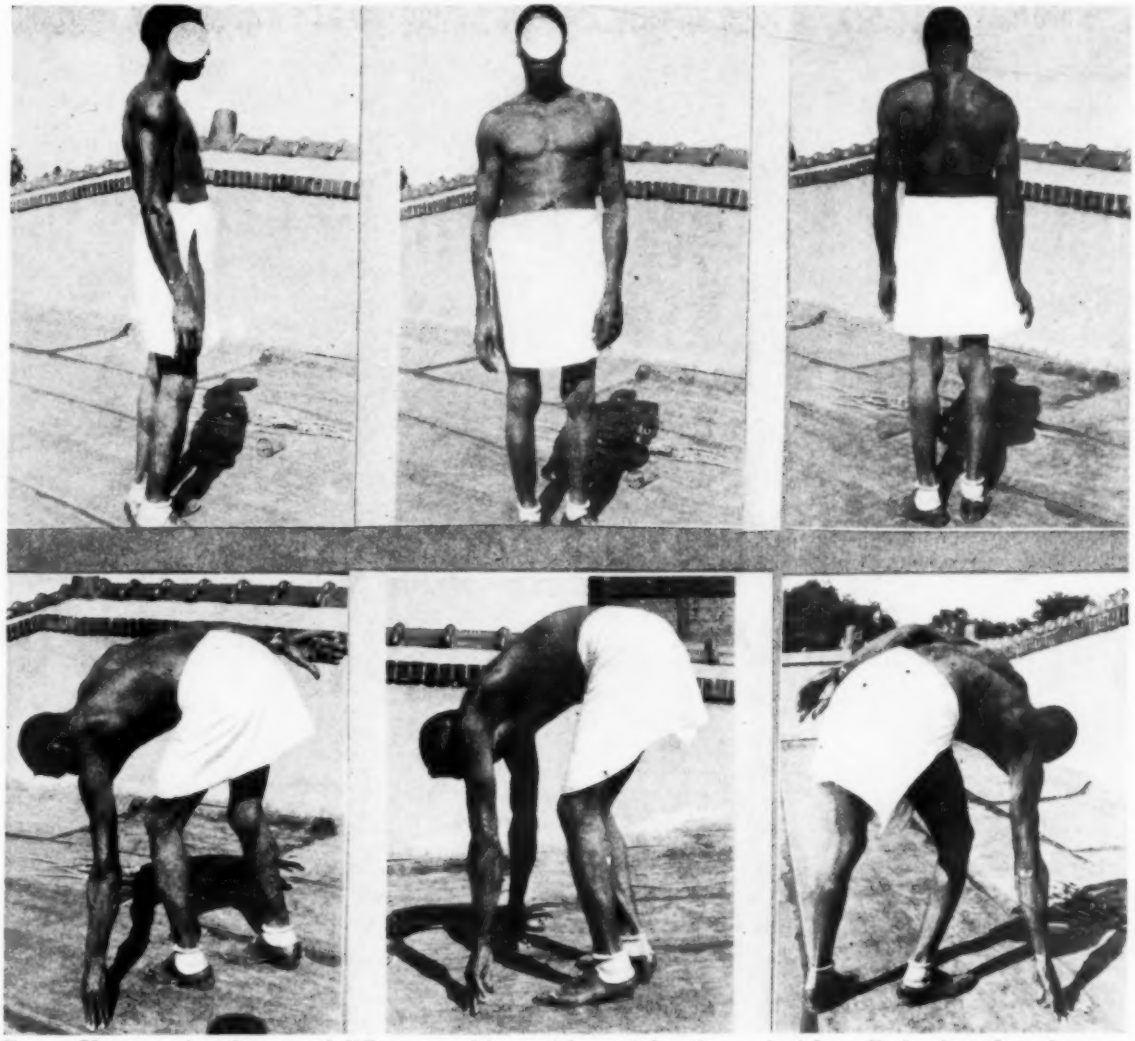


Fig. 3. Shows patient in several different positions without deformity and without limitation of motion two years after original fracture.

Operation: The operation was done under general anaesthesia. After making an incision about eight inches in length down to the vertebra, the following pathology was observed: there were many loose fragments of bone, the fragments being portions of the spinous process, the transverse process and the lamina of the second lumbar vertebra. These fragments were removed. It was necessary also to remove the posterior structure of the third lumbar vertebra to make room for reducing the cord. It was then observed that the body of the second lumbar vertebra above was split completely in half, longitudinally, and the entire vertebra above, with half the body of the second lumbar, was displaced laterally and downward about two inches. The cord was compressed and under considerable tension due to the lateral displacements of the vertebra, but was not severed nor was it badly damaged. After trying to reduce this fracture with moderate traction, which proved impossible, it was decided to remove enough of the bony structure of the vertebra to make a canal in order to replace the cord and prevent further tension and compression. This was accomplished and the wound closed. As stated above, it was impossible, because of the severe burns, to put this patient in any form of cast. He was,

therefore, placed in a Bradford frame and, as well as possible, moderate traction was made from above and below in order to immobilize

brace which he wore for three months. After being out of bed for two weeks this patient was allowed to walk around the hospital where he

Fig. 4. Shows fracture two years after original accident.

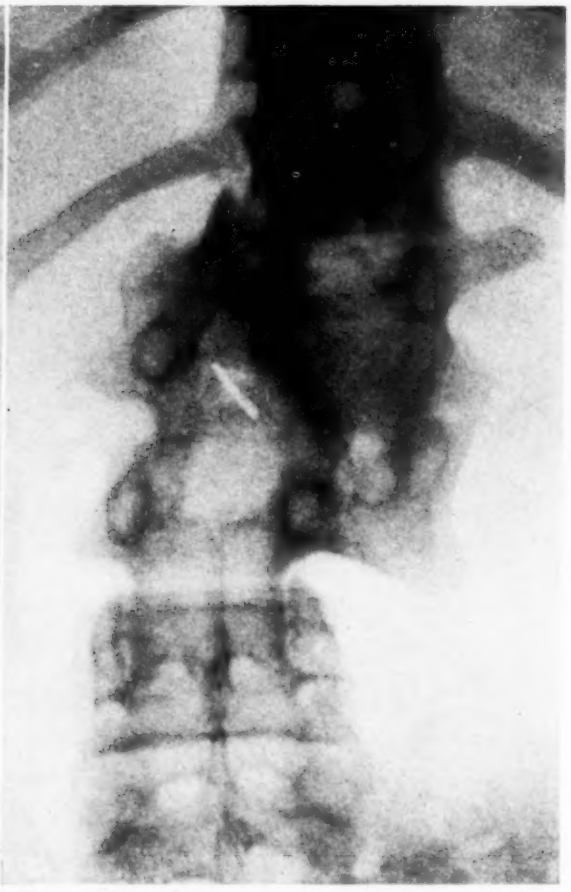


Fig. 5. Shows fracture seven years after original accident.

him. An *x*-ray was taken several days later. (See Fig. 2.) This shows all fragments of bone, posterior to the vertebra, removed and the canal where cord was replaced. It also shows that the fracture has not been reduced.

Progress while in hospital: Following the operation this patient showed no unusual symptoms. For the first few days he required catheterization. He suffered no unusual pain in the back. However, he complained a great deal of his burns. He also complained of some discomfort due to the extension used. One symptom which bothered him was the abdominal soreness and distention which are so often felt in fractures of the vertebra. The anesthesia in the lower extremities disappeared after four or five days.

This patient was kept in bed for eight weeks. He was then placed in a modified Taylor back

was observed for several weeks longer. During this time he was allowed to sleep without his brace, but he was required to wear it whenever in the standing or sitting positions. His recovery was uneventful and he was allowed to leave the hospital on July 6th, 1930, about fourteen weeks after injury.

Progress after leaving hospital: After leaving the hospital this patient wore his brace for two more months, finally discarding it without my permission. Also without my permission he drove an automobile several days after leaving the hospital, and eight months after injury secured a position as a truck driver. He has continued to work from this time and about two years following injury came to me for my opinion about getting a job doing manual work.

Condition two years after operation: This patient stated that he had no bad symptoms

since leaving the hospital, no anesthesia in lower extremities, no pains in the back, no stiffness in back, no limitation of movements of back in any direction. (See Fig. 3, A, B, C, D, E.) Observation showed no apparent deformity of back but his measurements showed a two inch shortening in his height. An *x*-ray was taken at this time which reported the following: an *x*-ray made, covering a period of two years, shows the original fracture and dislocation of the second and third lumbar vertebrae with marked lateral displacement and side-to-side union.

I advised that he could do any work desired at this time. Since then this man has done a laborer's duty and has missed no time from his work due to the injury. He has worked as a truck driver, saw-mill hand, brick yard laborer, common ditch laborer and at the present time, January, 1937—seven years after injury—is employed in a cotton compress where he moves 500 pound bales of cotton without difficulty and without any discomfort to himself.

CONCLUSION

My reasons for reporting this case are: (1) because of the severe nature of the fracture without permanent cord damage; (2) I believe by operating on this case immediately I prevented further cord damage due to pressure and tension on cord; (3) by removing the fragments of the lamina, spinous process and other posterior fragments of vertebra I prevented any eventual pressure of the cord due to callus formation, (4) I believe too great traction and manipulation in an effort to reduce this fracture without open operation would have produced permanent cord damage and, (5) from an industrial point of view, it shows that no matter how severe a vertebra may be fractured, there is not necessarily any permanent disability unless there is cord damage at the time of injury.

Hesselbach's hernia is a name derived from Franz Kaspar Hesselbach (1759–1816), a German surgeon.

Hodgkin's disease named after Thomas Hodgkin (1797–1866), an English physician.

Jacksonian epilepsy derives its name from John Hughlings Jackson (1834–1911), an English physician.

NECROSIS OF THE URINARY BLADDER*

WITH REPORT OF THREE CASES

JOSEPH TENENBAUM, M.D., AND SAMUEL SOIFER, M.D.

Attending Urologist, Central Neurological Hospital, Welfare Island; Associate Genito-Urinary Surgeon, Beth Israel Hospital

Adjunct Urologist, Beth Moses Hospital, Brooklyn; Assistant Urologist, Central Neurological Hospital, Welfare Island

NEW YORK CITY

THIS extremely serious and not infrequently fatal condition has not received the attention that it unquestionably merits. Up to 1934, O'Neil, Wolferth and Miller, Sterling and Hopkins, and others were able to collect about 300 cases from the literature. We believe, however, that gangrenous cystitis is a far more frequent complication than the number of publications would suggest. One of the reasons for the neglect to recognize the condition is the confusion in nomenclature. Cases representing the same pathological process and differing only in the degree of necrosis have been reported under such miscellaneous headings as: "Gangrene of the Bladder," "Exfoliative Cystitis," "Pseudomembranous and Diphtheritic Cystitis," "Cystitis Gangrenosa Dissecans," etc., thereby creating the false impression that we are dealing with several diverse clinical entities. Haultains' original suggestion to classify all these cases under the common denomination of "Necrosis of the Urinary Bladder" does away with unnecessary confusion and conforms more closely with the true pathology of the cases.

Etiology and Pathology of Bladder Necrosis. The etiological factors responsible for this condition can be classified in the following groups:

1. Poor nutrition of the urinary bladder wall (Haultains) caused by:
   a. Chronic urinary retention,
   b. Gravid impacted uterus,
2. Infection (O'Neil),
3. Chemical irritants,
4. Lesions of the central nervous system (cord bladder),
5. Mechanical obstructions,
6. Circulatory disturbances,
7. Metastatic necrosis.

Theoretically, each one of these factors may give rise to irreparable damage, resulting in gangrene. As a rule, more than one cause combines in creating a vicious cycle of retention, infection and circulatory disturbances. However, Heubner's and May's experiments suggest that the mechanical disturbances with impaired circulation are of greater importance in producing gangrene of the bladder than infection proper. The latter is particularly true in cases of gangrenous cystitis following transurethral resection of the prostate gland, which is becoming increasingly manifest as one of the possible complications of prostatic revision. It seems to us, however, that while the mechanical theory has been fully exploited, the metastatic factor has not been adequately emphasized. Its significance is borne out particularly in our first report.

In the great majority of cases of gangrenous cystitis, the destructive process is limited to the mucosa and submucosa. Where the destruction is more pronounced and extends to a greater depth, the muscularis and occasionally the serosa may likewise be involved. The various bladder layers then lose semblance to normal tissue and assume the characteristics of diffuse gangrenous exfoliation. The exfoliated membrane may separate in small shreds or

* From the Urological Departments of the Central Neurological Hospital, Welfare Island, New York, and Beth Moses Hospital, Brooklyn, New York.

be extruded *en masse*, presenting the appearance of a large cast of the bladder. In this respect it is similar to the pseudomembranous or croupous cystitis, the difference being only in the microscopic structure; the latter is composed of fibrin and cell detritus, while the true membrane contains also some bladder tissue. Such a clear-cut differentiation, however, is seldom possible on account of the rapid disintegration of the tissues involved. A further characteristic microscopic feature is diffuse cellular and bacterial infiltration with round cells, red cells and fibrin, separating the necrotic from the healthy zones.

With the separation of the necrosed tissue, and provided the patient survives, reparative changes begin and the bladder mucosa may even assume a normal appearance, although as a rule the viscus remains deformed and contracted.

Diagnosis. Bearing in mind that necrosis is the end product of deep embolic changes, and destruction of the bladder mucosa eventually reaching to the muscularis and subserosa, one can appreciate the difficulties in diagnosis in early cases, especially since the etiology comprises such a large variety of factors. We must adhere the more strictly to the few clues which the careful observer could hardly miss. The following points are of diagnostic importance:

1. The history of the case,
2. Urinary findings,
3. Cystoscopic investigation,
4. Microscopic examination of the exfoliated membrane.

Inasmuch as gangrenous cystitis is more apt to occur in people with hypertrophied prostate, urethral stricture, or bladder stone, and in pregnancies with impacted uterus or following bladder neck operations, the history of any one of these conditions should be carefully investigated. Any cystitis which, following cystoscopy or any other manipulation, becomes markedly aggravated, (especially in cases with a history of profuse hematuria, passing of pieces of membrane, and signs of toxemia), should arouse suspicion as to the possibility of incipient necrosis. Any attempt at catheterization at this time, if resulting in failure, is of diagnostic importance, since it may signify occlusion of the catheter with pieces of exfoliated membrane.

Cystoscopic examination, while often disappointing on account of marked hematuria and masses of loosely-attached membrane blurring vision, is nevertheless an important diagnostic adjuvant. In the early stages there is an acute, diffuse, hemorrhagic cystitis and, as the process progresses and ulceration sets in, the mucosa assumes a pale grey, dark grey, brownish or black color with bladder markings entirely obliterated. The surface may be covered with lime and other crystalline deposits. With the separation of the membrane, the bladder surface becomes shaggy and is covered with villous-like formation. An interesting observation noticed by cystoscopists is the impression of a large diverticulum. Actually, this is due to puckering of the dilated portion of the bladder after separation of the sloughed-off mucous membrane. A piece of membrane removed for microscopic study rarely fails to corroborate a well-founded preliminary diagnosis of bladder necrosis.

CASE REPORTS

Case 1. A. M., a white female nurse, age 40, born in the United States, was admitted to Central Neurological Hospital on January 5th, 1936, with the chief complaint of coughing, discharge from the ears, general weakness, and rise in temperature. Physical examination revealed the presence of broncho-pneumonia of the right lung and bilateral acute purulent otitis media. A smear taken from the throat and sputum yielded streptococci and pneumococci. On January 12th, the patient developed urinary difficulties with gross hematuria. Retention catheter was not tolerated. This condition continued until February 9th, when the patient developed complete urinary retention and bearing down pain in the suprapubic region. Catheterization was unsuccessful. After the administration of one ampule of pituitrin, a large irregular mass of greenish necrotic tissue having the appearance of a cast of the bladder was ex-

truded through the urethra, followed by a gush of some eighteen ounces of foul smelling urine.

Cystoscopy performed on the same day was extremely difficult on account of the presence of blood clots, large floating shreds and papillomatous-like masses. After thorough irrigation, the right ureteral orifice was located and catheterized. The right ureteral specimen contained a large number of red and white blood cells, single and clumped. The left ureteral orifice could not be visualized. Instead, there was on that side a large raw area extending to more than half of the bladder with a deep recess suggesting diverticulum. The right pyelogram showed the presence of moderate hydronephrosis with clubbing of the calices. Excretory urogram was inconclusive on account of poor visualization of the kidneys. Cystogram showed bulging of the bladder wall but no diverticula. Urine culture yielded streptococci. Blood culture was negative. Temperature throughout the course of the bladder ailment ranged between 100°F. and 102°F. Following the removal of the gangrenous membrane and cystoscopy, her general condition improved for a few days, followed by a renewed rise in temperature and gradual sinking, despite supportive measures. Patient expired on February 25th with evidence of severe toxemia and circulatory failure. Autopsy was not obtained.

Pathological examination of the extruded bladder case as reported by Dr. J. Rosenthal: Gross specimen (Fig. 1) consists of membranous tissue about 11 × 8 cm. and about 3 mm. in thickness, grey in color and of soft consistency.

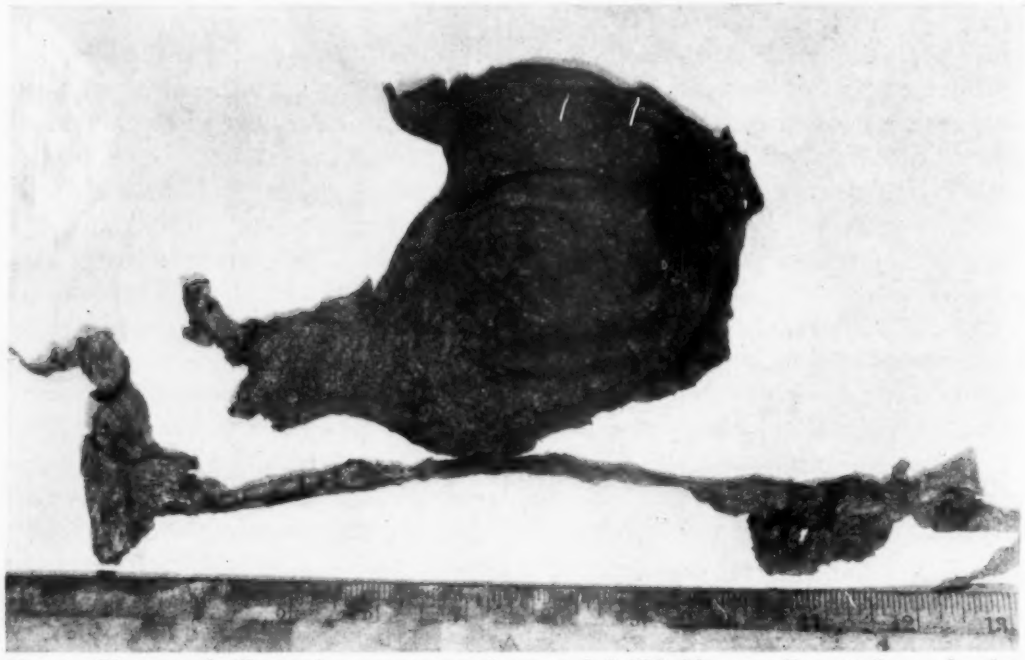


FIG. 1. Photograph illustrating spontaneously extruded "bladder cast," 11 × 8 cm. in size.

It is shaped in the form of a sac. The inner surface is irregular and hemorrhagic. The outer surface is covered by a shaggy layer of pinpoint nodules which resemble small deposits of fibrin. It is homogeneous throughout and shows no tissue differentiation.

Microscopic sections (Fig. 2): The tissue consists essentially of amorphous and necrotic material. There is considerable deposition of fibrin both on the outer and inner surfaces. There is only a relatively small amount of cellular material. Occasionally, areas are seen resembling necrotic muscle tissue.

COMMENT

We believe this to be a true case of exfoliative gangrenous cystitis of embolic origin, since streptococci had been recovered from the throat, discharging ears, sputum, urine, and subsequently rare groups of cocci suggestive of chain formation were found in the exfoliated membrane.

CASE II.* A. B., a white male, age 70, was admitted to Beth Moses Hospital on January

* This and the following case are reported from the service of Dr. N. Slutsky, Attending Urologist, Beth Moses Hospital.

21, 1936, with symptoms of prostatism. On January 28th, after one week of bladder drainage, the patient was subjected to transurethral resection of the prostate with a McCarthy Resectoscope. Eight pieces of tissue were removed. A retention catheter was kept in the bladder for two days. Following the removal of the catheter, the patient complained of severe tenesmus and hematuria. The temperature rose to 103°F. The catheter was replaced into the bladder and the patient given supportive measures. He expired on February 9, 1936.

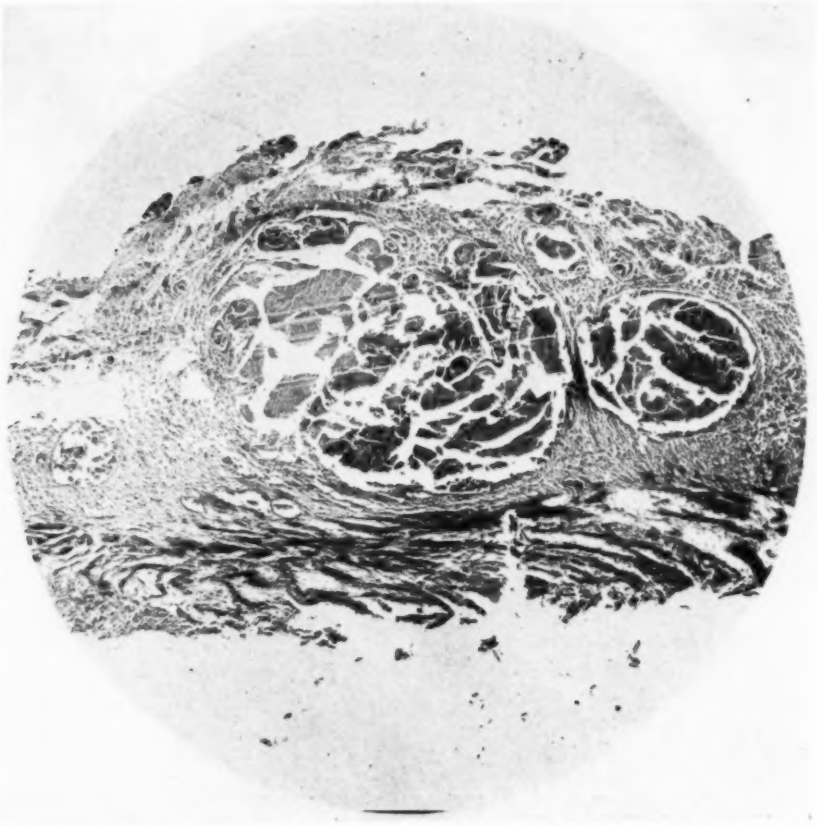


Fig. 2. Microphotograph of section of membrane showing necrotic mucosa with areas of necrotic muscle tissue.

Autopsy findings by Dr. A. Kantrowitz: Both kidney capsules strip with ease, revealing smooth regular surfaces, with cyanotic appearance on gross section and distinct cortical and medullary markings. Pelves and ureters present no gross changes. Bladder is markedly contracted. The wall is thickened to 1.5 cm. in some areas and shows marked ridge formation with sacculations and small diverticula. Mucosa reveals a marked hemorrhagic appearance and presents a variegated play of colors ranging from red to green and brown. At the trigone is a deep excavation with complete denudation of the mucosa. The area presents a shaggy, greenish-yellow exudate which forms the surface. A considerable portion of the median lobe had been removed previously and the surface is covered with the same greenish-yellow exudate which is present in the trigone. Both lateral lobes of the prostate show marked enlargement, together aggregating the size of a tangerine. The urethra shows no gross changes.

Microscopic findings (Fig. 3): The transitional epithelium is absent from large areas of the urinary bladder wall. There is considerable edema in the submucosa and superficial layers of the muscularis with polynuclear infiltration and necrosis of the tissues. The veins are markedly distended. The walls show collections of polynuclear leucocytes with thrombi adherent to the walls of the vessels.

Case III. S. H., a white male, age 70, was admitted to Beth Moses Hospital on January 31, 1936 with symptoms of retention, dysuria and urgency. He had been catheterized daily for the past ten days. On February 3rd, after

five days of continuous drainage, transurethral resection of the prostate was performed, and fourteen pieces of tissue removed. Despite immediately distal to the verumontanum leading into the bladder along the mesial aspect of the right lobe.

FIG. 3. Microphotograph illustrating absence of transitional epithelium; edema and polymorphonuclear infiltration of submucosa and muscularis; and areas of necrosis. Note large thrombus in vein.

retention catheter, the patient continued to have tenesmus and hematuria. He expired on February 8, with signs of severe toxemia.

Autopsy findings: Both kidney capsules strip with ease, revealing a number of fine, dome-shaped protuberances. On puncturing these, clear fluid was obtained. Bladder is markedly contracted. The wall, measuring up to 2 cm. thickness in some areas, is firm and rigid. The mucosa presents a variegated play of colors, ranging from red to black, with a greenish-black discoloration on the surface of the rugae. The trigone and vesical neck reveal the same greenish-yellow shaggy appearance, with an exudate forming the base of the otherwise denuded surface. Considerable tissue appears to have been removed from the surface of the median lobe and from the area immediately proximal to it, resulting in the formation of a deep excavation in the trigone. The urethral channel in the prostatic portion appears to have been shunted to the left, with the formation of a false urethral passage from an area

Microscopic findings: The transitional epithelium is absent in numerous places. The submucosa and muscularis show marked edema, with polynuclear infiltration and areas of necrosis, obliterating the normal architecture of the bladder wall.

Discussion of Cases 2 and 3: The apparent factor in the causation of the gangrene in these two cases was the result of interference with the blood supply of the bladder wall, either through too deep a coagulation necrosis or to the resection of too extensive an area of the bladder mucosa. Infection, we believe, played a minor role, since these patients had been cystoscoped and drained by catheter over a long period of time, with the production of the usual cystitis. Why the operative procedure should produce a gangrenous cystitis can be explained by interference with the blood supply which, coming mainly from the

superior vesical, inferior vesical, and inferior hemorrhoidal arteries, enters the bladder at its base and then spreads out fanwise to supply the rest of the bladder wall. Thrombosis of these important blood vessels is ample ground to account for the extensive necrosis. The prognosis in such cases depends on the severity of infection, the degree of accomplished necrosis, and the early recognition of the condition. As a general rule, bladder necrosis is more grave in men than in women (75 per cent in men to 50 per cent in women, according to Wolferth and Miller). It is invariably fatal when occurring as a terminal affair in cases with debilitating disease.

It is of the utmost importance in cases subjected to prostatic resection, not to be overzealous in the coagulation of bleeding points and to avoid extensive injury to the mucous membrane. The presence of devitalized tissue, especially when part of the trigone has been injured, favors the spread of infection and gangrene. Moreover, the best performed prostatic resection leaves a great deal of edema and infiltration at the bladder neck, causing obstruction and interfering with drainage for days and sometimes weeks after the operation. During all this time, the bladder is filled with infected residual urine and decomposed necrotic tissue, which may result in irreparable damage to the bladder wall. A similar condition prevails in cases where the retention catheter becomes obstructed or, due to improper insertion, fails to keep the bladder free from residual urine. Post-operative management of the cases, it seems to us, is even more important in these cases than the operative procedure itself.

Where necrosis has set in, early removal of the membrane permitting free drainage is of paramount importance. In the male, because of anatomic difficulties, suprapubic cystotomy for the removal of necrotic tissue and free drainage is indicated. This point has been particularly stressed by Wolferth and Miller, who have shown that there is a greater percentage of deaths in the cases where this precaution was neglected.

SUMMARY AND CONCLUSION

Three cases of necrosis of the urinary bladder are added to the growing literature on the subject.

The first of our reported cases is especially interesting as an illustration of the metastatic etiology of necrosis due to bacterial emboli of the bladder wall following an ear and lung condition, all of them caused by a streptococcus infection.

The other two cases are the result of transurethral resection of the prostate, which factor in the production of bladder necrosis is becoming more and more evident.

REFERENCES

1. Schüppel, A. *Arch. f. Klin. Chir.*, 145: 380. 1927.
2. Heidler. *Wien. Klin. Wochenschr.*, 40: 571 (April 28) 1927.
3. Patch, F. S. *J. Urol.*, 19: 713. 1928.
4. O'Neil, R. F. *Tr. Amer. Assoc. Genito-Urin. Surgeons*, 4: 282. 1909.
5. Giesecke, H. *Zeitschr. f. Urol.*, 25: 561. 1931.
6. Duchanoff, A. *Ztschr. f. Urol. Chir.*, 29: 516. 1930.
7. Maluschew, D. *Deutsche Med. Wochenschr.*, 55: 794 (May 10) 1929.
8. Lindemann, W. *Zentralbl. f. Gynäk.*, 55: 3582 (December 12) 1931.
9. Stirling, W. C. and Hopkins, C. A. *Jour. Urol.*, 31: 517. 1934.
10. Wolferth, C. C. and Miller, T. G. *Am. J. M. Sc.* clxvii: 339. 1924.
11. Cowen, L. B. and Cowen, R. *Urol. and Cutan. Rev.*, 37: 105 (February) 1933.
12. Kraas, E. *Arch. f. Klin. Chir.*, 183: 595. 1935.

FRACTURES OF FIRST RIB UNASSOCIATED WITH FRACTURES OF OTHER RIBS*

FRANK J. BRESLIN, M.D.

LOS ANGELES, CALIFORNIA

FRACTURES of the first rib alone are surprisingly rare. A thorough search of American and foreign literature reveals a chaotic condition and only 27 reported cases from twenty-two authors. It is the opinion of the author that bringing the literature to date, and the report of five additional cases, might be of interest.

The first rib undoubtedly has been fractured on various occasions and the condition overlooked because of associated pathology sufficient in itself to cause death. But it is difficult to comprehend why this interesting, and at times serious clinical entity, has been reported but twenty-seven times in the medical literature. For the past twenty-five years, surgeons caring for traumatic cases, have resorted to the use of the Roentgen ray and literally millions of roentgenological examinations have been made of the human chest, but very few diagnoses of this condition have been reported. Many surgeons have seen the bony cage fractured and compressed, and at times, have seen the first rib fractured in these instances, but fractures of the first rib, alone, have been few and have been set down in some textbooks as the rarest of all fractures.

ANATOMY

The first rib is situated deeply in the neck. It is the shortest of all the ribs, is flat and broad having two surfaces which face upward and downward. The upper surface has two shallow depressions separated by the *tuberculum scaleni* to which is attached the scalenus anticus muscle. Over the groove in front of the *tuberculum* passes the subclavian vein, behind it passes the subclavian artery. The scalenus medius muscle is attached to a rough spot between the groove for the subclavian artery and the tuberosity. The outer border of the rib is convex and at its posterior portion the first serration of the serratus magnus is attached. The first rib therefore, has three muscles attached to it. It articulates with the first thoracic vertebra posteriorly and through its costal cartilage with the sternum anteriorly.

MECHANISM OF FRACTURE

The first rib is found approximately in the center of the neck and it is adequately protected by the clavicle anteriorly, the scapula posteriorly together with the muscles of the shoulder girdle. It is difficult to comprehend how direct trauma can reach the rib itself. We may therefore presume that fracture must be caused by indirect or transmitted violence. Johnston[7] states,

> Mr. Lane, who has given great attention to this subject both in examination of dissecting-room subjects and in production of artificial fractures in the cadaver, says that fracture of the first rib can take place only in one or other of three ways: (a) indirect violence, the force being transmitted through the clavicle; (b) directly by means of force applied behind; (c) indirectly, force transmitted through manubrium.

PATHOLOGY AND COMPLICATIONS

The first rib may be fractured anywhere from its costal cartilage to the spine. The fracture line may vary from a simple fissure to complete comminution. Healing is the rule but pseudarthrosis is to be considered. Complications most frequently reported are (1) abscess formation above or below the clavicle, (2) rupture of the apex of the lung, (3) empyema, (4) pleurisy, (5) hemorrhage, and (6) nerve symptoms, which may come on immediately or a few months

* From the Surgical Service of St. Vincent's Hospital.

after the accident when the callus is of sufficient size to encroach upon the nerve pathways.

SYMPTOMS

The symptoms encountered depend upon the location and severity of concomitant pathology. Nearly all patients complain of a "sticking pain" in the region of the shoulder. There may or may not be pain located at the site of the fracture. Movement of the arm, head and neck will cause pain in the region of the fracture and this may be explained by muscular pull. Dyspnea is not great unless there has been a rupture of the pleura or of the lung. Numbness or tingling in the arm of the affected side is not usually present unless the fracture is complicated by a laceration of a nerve pathway, early, or later on by impingement of growing callus upon certain nerves. Other symptoms which might be enumerated are in reality complications.

TREATMENT

If we do not encounter severe complications involving deeper structures, the method of choice is immobilization. This can be adequately maintained by adhesive plaster strapping applied to the chest. Immobilization of the lower ribs will satisfactorily fix the first rib. With an assistant holding the patient's arms above his head, the strapping is applied from above downward and nearly encircling the entire chest. Anteriorly the straps may be applied in an oblique manner. Damaging pressure is prevented by using a generous pad in the axilla. The forearm and arm of the affected side are held close to the patient's chest by means of a sling. Pain is usually relieved immediately and the period of treatment is approximately four weeks.

CASE REPORTS

Minne[21] in 1932 stated that, excepting his case, there was not a report in the literature of a child suffering with a fracture of the first rib and but few of adults. The first case reported was that of Jones, in 1869. We will present the other cases in chronological order.

Case I. (Jones,[1] 1869.) A male, aged 50 years, fell while in a street fight, striking the right side of his neck on the curbing. Immediately, his adversary kicked him several times in the neck and death ensued in a few minutes. Autopsy revealed a fracture of the right first rib, close to the spine, with a piece of the rib, two inches in length, detached and lying below the clavicle and embedded in the upper part of the lung. The right subclavian artery and brachiocephalic vein were torn through and massive hemorrhage and emphysema were encountered. Jones was of the opinion that the fracture was caused by the fall and that the detachment and separation of the fractured fragment was due to the subsequent kicking of the patient's neck.

Case II. (Lyell,[2] shortly previous to 1886.) In Holmes' "System of Surgery," Lyell reported about a child who had been run over, and who lived but a few hours after the accident. Autopsy showed a fracture of the left first rib near its neck, together with a laceration of the pleura and a small rupture of the lung.

Case III. (Hunter,[3] 1886.) In the museum of the Royal College of Surgeons, in England, Hunter labeled one case of fracture of the first rib.

Case IV. (Lane,[4] 1887.) Sir Arbuthnot Lane recorded four cases from the dissecting room of Guy's Hospital. All of his cases were found on the right side.

Case V. Morton,[5] (1893) reported a case for Mr. C. F. Pickering, of Bristol. A man, aged 60 years, fell on the right side of his body, fracturing his right arm. One week later, an abscess of the right sternoclavicular articulation was incised and twenty-six ounces of pus evacuated. Autopsy showed two fractures of the first rib, not diagnosed during life.

Case VI. Bahr[6] (1894) quotes a case of Linder of a fracture of the right first rib caused by the recoil of a gun. There were no untoward symptoms.

Case VII. (Johnston,[7] 1896.) A female, aged 45 years, while carrying a fender fell and sustained a compound fracture of her left ring finger. Nine days later she was admitted to the hospital because of a tender swelling over the left first intercostal space. Later she coughed up pus and shortly afterwards an incision was

made over the second left interspace and an abscess was encountered together with a comminuted fracture of the first rib. The patient was discharged cured fourteen days later.

Case viii. Chudowszky,[8] (1898) mentions a case of fracture of the first rib, right and left. There was no adequate case history other than that the patient fell, sustaining the fractures.

Case ix. (Bennett,[9] 1905.) A male, aged 57 years, while unloading sacks of wheat, fell backwards some three feet, sustaining a fracture of the cartilage of the left first rib, also a fracture of the left clavicle between the conoid and trapezoid ligaments. He made a satisfactory recovery and stated very accurately that a direct blow did not strike his collar bone or left chest wall.

Case x. (Codd,[10] 1916.) A female, aged 45 years, was in an automobile collision and complained of pain in the right arm. Roentgen ray examination revealed a fracture of the right first rib. This patient made a satisfactory recovery.

Case xi. (Milligan and Ford,[11] 1921.) A female, aged 17 years, complained of a swelling on the right side of her neck. She had "pins and needles" and a cramp-like sensation in the right arm and hand. The history revealed that the patient had fallen down stairs two months previously and examination demonstrated a bony swelling, about the size of a walnut, one inch above the clavicle. This swelling was continuous with the first rib. The diagnosis of fracture was confirmed by a roentgen ray examination.

Case xii. (Payne,[12] 1923.) A man, aged 40 years, was in an automobile wreck and when found the wheel of the automobile was resting upon the patient's chest. There was considerable tenderness over the right first and second ribs. Roentgen ray examination revealed a fracture of the first rib through the cartilaginous portion close to the sternum. The patient was discharged to his regular occupation, that of a railway conductor, at the end of six weeks.

Case xiii. (Ameuille,[13] 1925.) A female, aged 68 years, awakened during the night suffering with pain over the right first rib area. When examined there was a swelling near the anterior end of the first rib, which swelling persisted for several months. Roentgen ray examination showed a complete fracture of the costal cartilage. The cause of the fracture was never determined. Recovery was uneventful.

Case xiv. Wahl,[14] (1926) reports two cases of spontaneous fracture of the first rib, probably those of Ameuille and Libensky. The first case was that of a female, aged sixty-eight years, who sustained a fracture of the right first rib after trauma. The second case was that of a male, aged thirty-six years, who sustained a double fracture of the left first rib after an accident. There were no untoward symptoms.

Case xv. (Pohl,[15] 1929.) A female, aged 16 years, fell to the ground in a kneeling position. She later felt a stabbing pain in the region of the right shoulder. Roentgen ray examination showed a complete fracture of the right first rib at about its middle. There was an uneventful recovery.

Case xvi. (Ostrowski,[16] 1930.) A male, aged 26 years, met with a street accident, losing both of his legs. Four weeks later an operation was performed revealing a fracture of the right first rib, a rupture of the right subclavian artery, together with a rupture of the upper portion of the right lung. The patient died of a hemorrhage from the lung.

Case xvii. (Porzelt,[17] 1931.) An adult male had his right arm stretched out carrying a weight when he felt a violent sticking pain in the right shoulder region. Eight days later there was demonstrated a right upper pleuritis and roentgen ray examination showed a transverse fracture of the right first rib. The author was of the opinion that this fracture was the result of muscular pull. The patient made a satisfactory recovery.

Case xviii. Werner,[18] (1931) reported an isolated fracture of the first rib in a male, forty years of age. The details were clouded but Werner thought that the etiological factor was muscular pull. Convalescence was uneventful.

Case xix. Pittowa,[19] (1932) cites two patients who were subjected to roentgen ray examinations for various causes and fractures of the first rib were found. One was a male, aged 36 years, and the other was a female, aged 16 years. Both of these patients had had a "sticky pain" in the region of the right shoulder. It was difficult however to set a definite time as to the onset of causative trauma.

Case xx. (Adler,[20] 1932.) A male laborer, aged 38 years, while raising a heavy burden, felt a severe "sticking pain" in the region of the right shoulder. Roentgen ray examination revealed a transverse fracture of the right first rib. The patient made an uneventful recovery.

Case xxi. (Minne,[21] 1932.) A child, aged 6 years, after a fall presented a fracture at the junction of the anterior and middle third of the

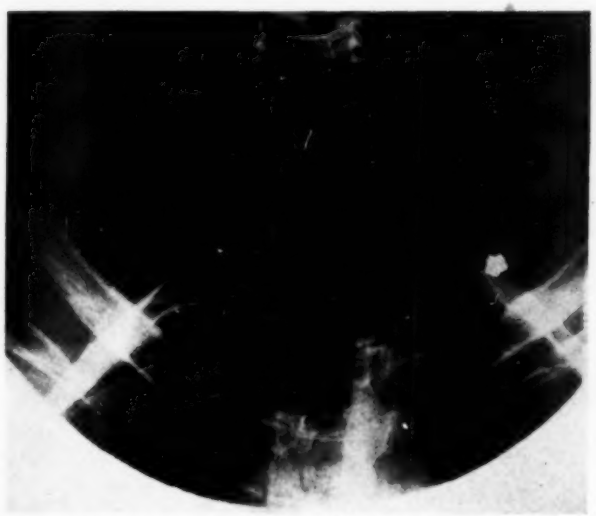


Fig. 1. (Case xxiii.) Fracture of the right first rib near the spine.

right first rib. Minné said that there was no other case in the literature of a child, and but few cases of adults. The patient made a satisfactory recovery.

Case xxii. (Rebhorn,[22] 1933.) A male, aged 21 years, sustained a squeezing violence to the upper part of the left chest. He demonstrated a fracture of the left first rib together with the left scapula. Seven weeks later the patient presented himself for treatment and demonstrated Horner's syndrome. The fractures healed but the eye symptoms apparently persisted.

AUTHOR'S CASE REPORTS

Case xxiii. (1935.) A male, aged 56 years, fell twenty-two feet from a tank, striking on his side with his neck flexed toward the right. There was a momentary period of unconsciousness followed by pain over the right side of the neck, accompanied by pain in the root of the neck and the right side of the pelvis. Roentgen ray examination disclosed a fracture of the right first rib near the spine together with a fracture of the transverse processes of the fifth, sixth and seventh cervical vertebras on the right side (Fig. 1). There was also a fracture of the wing of the right ilium.

The patient's neck was placed in one pound halter traction and the chest was immobilized with adhesive plaster strapping. The patient walked out of the hospital seven weeks after the accident and made a satisfactory recovery.

Case xxiv. (1935.) A male aged 50 years,

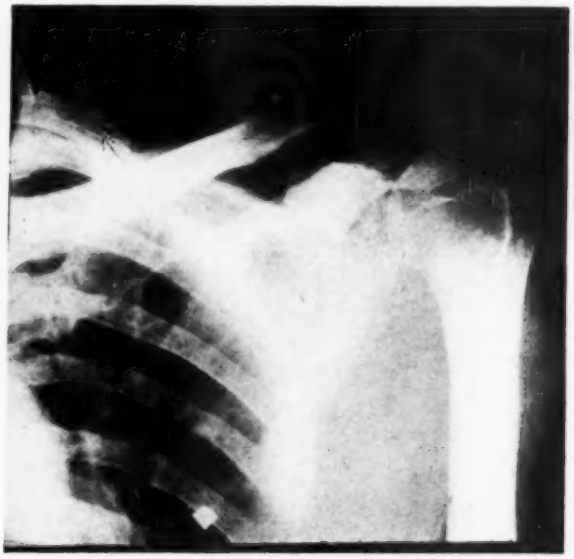


Fig. 2. (Case xxiv.) Fracture of the left first rib near the spine.

who while inside a building, was struck over the top of the left shoulder by a falling beam. Immediately a "sticking pain" was noticed in the left shoulder region. Roentgen ray examination disclosed a fracture of the left first rib near the spine (Fig. 2). The chest was immobilized by means of adhesive plaster strapping with immediate relief. The patient remained in the hospital but three days, rested at his home for one week, and then went to his office. Six weeks after the accident, he was without subjective complaints.

Case xxv. (1935.) A male, 21 years of age, was in an automobile wreck turning over three times, sustaining painful injuries in and about his right shoulder region. He was admitted to the hospital two and one-half days later and roentgen ray examination disclosed a comminuted fracture of the right scapula together with a fracture of the first rib one and one-half inches lateral to the cervical spine (Fig. 3). The right chest wall and scapula were immobilized with adhesive tape and the patient left the hospital thirteen days later, to place himself under the care of a doctor in a nearby town. We have no further record of the final result of this patient.

Case xxvi. (1935.) A male, 39 years of age, was in an automobile collision striking the front of his face and the anterior chest wall against

the steering wheel. When admitted to the hospital, he complained of pain in the upper anterior aspect of the left chest wall. Roentgen

Fig. 3. (Case xxv.) Fracture of the right first rib 1½ inches lateral to the cervical spine.

ray examination disclosed a complete fracture 1½ inches long over the center of the left first rib (Fig. 4). The patient's chest was immobilized

Fig. 5. (Case xxvii.) Fracture of the left first rib at the region of the junction of the rib with the cartilage.

with adhesive tape and one month later, he was discharged from the hospital and at the end of another week, was discharged to his work, that of a machinist. His recovery was complete and without complications.

Case xxvii. (1935.) A male, 60 years of age, was knocked down and struck by the right front fender of an automobile and thrown so

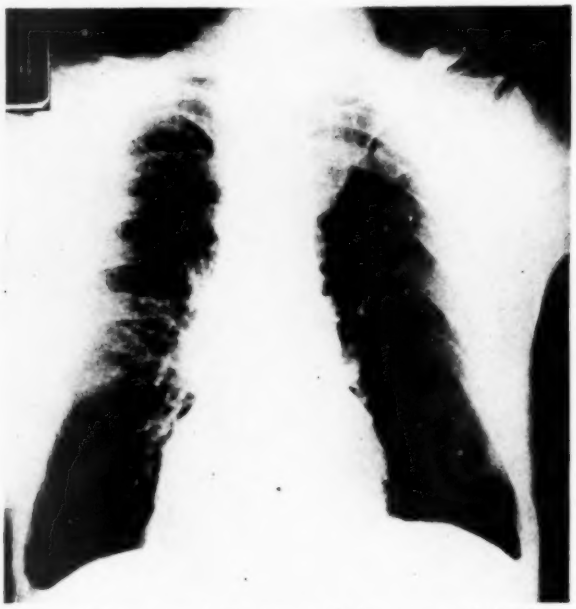


Fig. 4. (Case xxvi.) Fracture of the center of the left first rib.

that his chest wall, at about the region of the left nipple, struck the curbing. He was not unconscious and walked away. Twelve hours later he noticed pain in the region of the left chest region. At this time he was admitted to the hospital and Roentgen ray examination disclosed a complete fracture of the left first rib at the region of the junction of the rib, with the cartilage (Fig. 5). The chest was immobilized with adhesive tape and six weeks after the accident, he was released to his work, that of a cabinet maker, practically free from symptoms. The patient had no complications and his recovery was complete.

CONCLUSIONS

Fractures of the first rib, although rare, are to be considered in patients who complain of a "sticking pain" in and around the shoulder region, particularly in those patients, who after severe trauma, do not show signs of fracture of the neck, clavicle or humerus.

A first rib fracture is usually associated with a contusion or laceration of the pleura, or an extravasation of blood, of greater or lesser degree, from the subclavian vein or one of the smaller veins.

It is important to bear these conditions in mind and treat the patient in a hospital thereby obviating the possibility of emphysema, abscess formation or delayed secondary hemorrhage.

The author wishes to acknowledge a debt of gratitude to Dr. K. S. Davis, Roentgenologist to St. Vincent's Hospital, also to Dr. R. J. Flamson, on whose service Cases XXV, XXVI and XXVII were treated.

REFERENCES

1. Jones, T. E. *Brit. Med. J.*, 2: 201, 1869.
2. Lyell, R. W. Quoted by G. Jameson Johnston. *Dublin J. M. Sc.*, 102: 194 (September 1) 1896.
3. Hunter, Mr. Quoted in Holmes's Syst. *Surg.*, 1: 749, 1881.
4. Lane, W. Arbuthnot. *Brit. Med. J.*, 2: 119 (July 16) 1887. Phila., Lea & Sons.
5. Morton, M. *Brit. Med. J.*, 1: 406 (February 25) 1893.
6. Bahr, (Linder) F. *Dtsch. z. chir.*, 39: 251–280, 1894.
7. Johnston, G. Jameson. *Dublin J. Med. Sc.*, 102: 191–196 (September 1) 1896.
8. Chudowszky, M. *Beitr. z. klin. Chir.*, 22: 643–728, 1898.
9. Bennett, E. H. *Dublin J. Med. Sc.*, 120: 81–84, 1905.
10. Codd, T. P. *Med. Pres. and Circ.*, 101 n.s. 196 (March 1) 1916.
11. Milligan, E. T. C. and Ford, R. E. *Brit. Med. J.*, 1: 191 (Feb. 5) 1921.
12. Payne, M. I. *Internat. J. Surg.*, 36: 113–116 (March) 1923.
13. Ameuille, P. *Bull. et. Mem. Soc. Med. de Hop. de Par.*, 49: 147–149 (January 30) 1925.
14. Wahl, E. *Wien. klin. Wchnschr.*, 39: 1213–1217, 1926.
15. Pohl, R. *Med. klin.*, 25: 636–637 (April 19) 1929.
16. Ostrowski, S. *Zentralbl. f. Chir.*, 57: 745, 1930.
17. Porzelt, W. *Zentralbl. f. Chir.*, 58: 1264–1267, 1931.
18. Werner, H. Zitient Zbl. Radiol. Hft. *Dtsch. z. Chir.*, 23: 12, 1931.
19. Frank-Pittowa, H. *Roentenpraxis*, 4: 1011–1013 (December) 1932.
20. Adler, A. *Zentral. f. Chir.*, 59: 518–521.
21. Minne, J. *Rev. d'orthop.*, 19: 25–37 (January) 1932.
22. Rebhorn, E. H. *Bull. Moses Taylor Hosp.*, Scranton, Pa., 2: 2, 1933.

Ludwig's angina is a purulent inflammation seated around the submaxillary gland, named after Wilhelm Friedrich von Ludwig (1790–1865) a German surgeon.

Mikulicz's disease (achro-acytosis) is named after the Polish surgeon, Johannes von Mikulicz-Radecki (1850–1905).

Pott's disease, caries of the vertebrae, is named after Percival Pott the English surgeon (1714–1788).

A NEW COMBINATION T-TUBE AND DUODENAL CATHETER FOR CHOLEDOCHOSTOMY*

ABRAHAM STRAUSS, M.D., F.A.C.S.

CLEVELAND, OHIO

IT has been common practice for many years to drain the common bile duct after removal of stones or for infection;

FIG. 1. (Mrs. S., August 4, 1936.) Drainage tube with the catheter ready for use.

and to drain from two to eight weeks, depending on the lesion. During the drainage no bile passes into the intestinal tract unless fed to the patient or when pressure is instituted by clamping the T-tube for several hours. Bile is hard to disguise, so that it cannot be taken per os unless given in capsules and a great number of capsules are necessary to give any beneficial amount of bile.

L. MacArthur long ago advocated the use of the catheter placed through the choledochus into the duodenum to serve as a means of administering fluid immediately after operation. Thus, with two catheters in the choledochus, one to drain, the other to pass into the duodenum, bile itself could be collected and then poured through the tube into the duodenum.

Dr. D. Foldes, in a personal communication several years ago, suggested that T-tubes should be manufactured with a division in the long upright part of the T-tube through which a catheter could be passed to accomplish this.

The patient, Y. S., a female, age 45, was admitted to Mount Sinai Hospital on July 13, 1936, complaining of pain in the right upper quadrant and fever.

On January 17, 1936, Dr. L. Krauss had performed a cholecystectomy and choledochostomy for chronic and acute cholecystitis, with cholelithiasis. Many stones were removed from the common duct, which was then probed and found patent. A T-tube was left in for drainage. Recovery was uneventful—the tube was removed after eight weeks.

On March 20, she was readmitted to the hospital because of pain in the right upper quadrant, fever and chills. Her temperature was 103.4 but became normal the next day and remained so. She was not jaundiced. Her icteric index was 5, the Van den Bergh 0.2. Her urine was negative. White count on admission was 17,800. She was discharged well from the hospital on the fourth day.

On July 4, 1936, she was again admitted with the same complaint and the same finding, and discharged well on July 10.

She was admitted again three days later with a recurrence of symptoms and now for the first time the icteric index was elevated, 13, and the Van den Bergh was 2.0. The latter was 0.2 on July 16, hydrobilirubin was present in the stool and her temperature was normal. The pre-operative diagnosis was cholangitis with possibly a stone in the common duct, so I operated on her on July 17th. It was at this time that I used the new type of tube illustrated in Figure 1. I found a dilated common duct with some

* From the Surgical Service, Mt. Sinai Hospital, Cleveland, Ohio.

fine gravel and infection. I decided to modify the T-tube by placing a catheter through it into the duodenum and letting the external end of the catheter come out through a hole I made in the side of the tube. Thus I was enabled to collect bile from the patient and reinject it through the catheter.

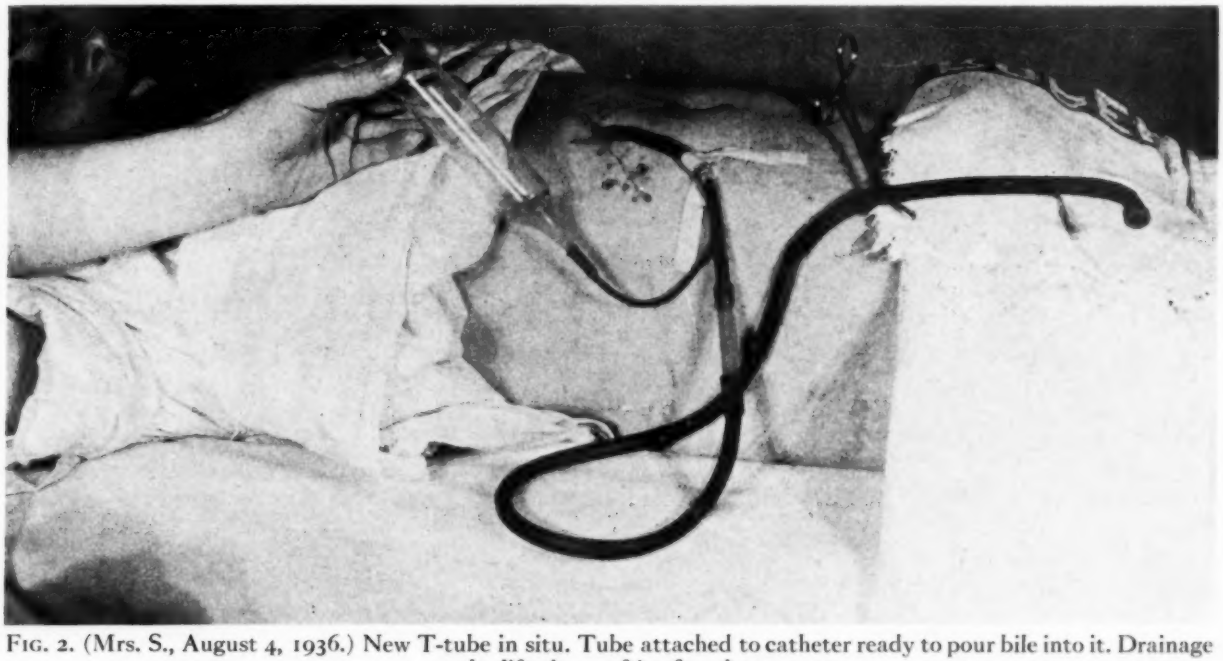


Fig. 2. (Mrs. S., August 4, 1936.) New T-tube in situ. Tube attached to catheter ready to pour bile into it. Drainage tube lifted out of jar for photo.

On the day after operation, three ounces of 5 per cent glucose was poured through the catheter by gravity every two hours. The following day the bile was clear enough to be fed through the catheter, so 3 ounces of her own bile was poured through the catheter twice a day. This was continued until August 10th, when the catheter was removed and the patient was discharged from the hospital. Thus, the catheter was of service for the administration of bile for three weeks. The T-tube was left in for a total of eight weeks and was clamped at intervals for several hours at a time. The wound healed promptly when the tube was removed.

Since then, I used the tube on one other patient and Dr. Howard Gans has used this tube on two of his patients. He used the catheter to instil saline, then glucose into the duodenum and on the fifth day post-operative began instilling one ounce of bile twice a day. He removed the catheter on the seventh day and the tube on the fourteenth day postoperative.

On his second case he administered 300–400 cc. of bile a day through the catheter from the third day post-operative for four days. He removed the catheter on the eighth day post-operative, and the tube on the sixteenth day.

Thus, such a tube serves the double purpose of feeding saline or glucose to a patient immediately post-operative, and bile as long as one wishes to leave the tube in place.

NEW INSTRUMENTS

INTESTINAL SEWING CLAMPS

THEIR USES AND ADVANTAGES*†

HANS MAY, M.D.

PHILADELPHIA, PENNSYLVANIA

IN most gastro-intestinal operations, the stomach or the gut, or both, are opened temporarily. Since the establishment of modern surgery, many attempts have been made to change the open procedure into a closed method. All kinds of instruments have been devised for that purpose. One of these is the Petz sewing clamp and its modifications. The principles involved in the use of these instruments are, first, crushing the line of intended section, and second, inserting a double row of tiny metal clips across the crushed line. Such a suture makes the operation quick, safe, simple and aseptic.

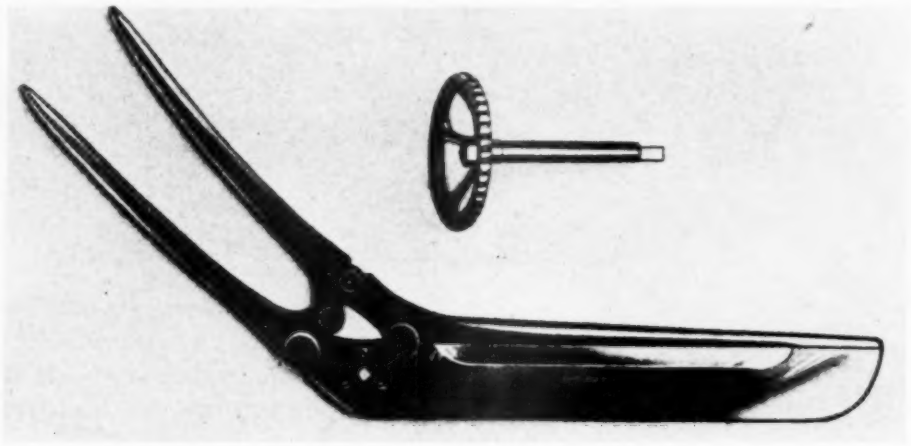


FIG. 1. Intestinal sewing clamp after v. Petz.

The original v. Petz clamp (Fig. 1) resembles Payr's crushing clamp. The upper part contains the stitching mechanism, which, before use, is loaded with fine new-silver hooks. The stomach or gut to be sutured is placed between the opened jaws of the clamp, the forceps is then closed, thereby crushing the organ. The turning of a wheel withdraws a bar furnished with teeth which presses the tiny hooks into and through the tissue. The clamp is now opened and removed. The organ is separated with straight scissors or electric knife at a place between the two straight rows of fine stitches situated at both sides of the crushed furrow. Thus the organ is closed in a few seconds in an aseptic and water-tight manner both proximally and distally. (Fig. 9.) The clips are buried by an interrupted or continuous Lembert suture.

The original v. Petz sewing clamp, however, has some disadvantages, as it is not curved at the right angle and is too large. I have found that it could not be used at certain places in the abdominal cavity, for instance, deep down in the pelvis or high up under the costal margin; and these are

* From the Surgical Service B of the Lankenau Hospital.
† Read before the Philadelphia Academy of Surgery, No. 2, 1936.

just the places where a mechanical suture would be a great convenience. Another disadvantage of this machine is, that it must be reloaded between each application. Although there is now a smaller Petz clamp available, the rest of the disadvantages remain.

A welcome modification (after Neuffer) appeared recently. (Fig. 2.) The main advantage of this clamp is that it carries the clips in separate magazines and two or more sutures can be applied by simply exchanging one empty clip magazine for another already filled. After charging the magazine and fitting it correctly in position, the stomach or intestine to be sutured is brought into the space between the open jaws. A firm but not too forcible pressure of the gripping handles will compress the grasped parts. The pressure is automatically maintained by the stop lever (2). By reversing the lever (1) in the longitudinal direction, the rack below is set free. By a further continuation of the manual pressure on the handles, the clips in the magazine are pushed forward. The suture being applied, the pressure on the handles is released and the instrument removed. The further manipulation of the sutured organ is the same as already described.

part to be sutured is brought between the opened branches of the forceps. A firm pressure on the handles at first compresses

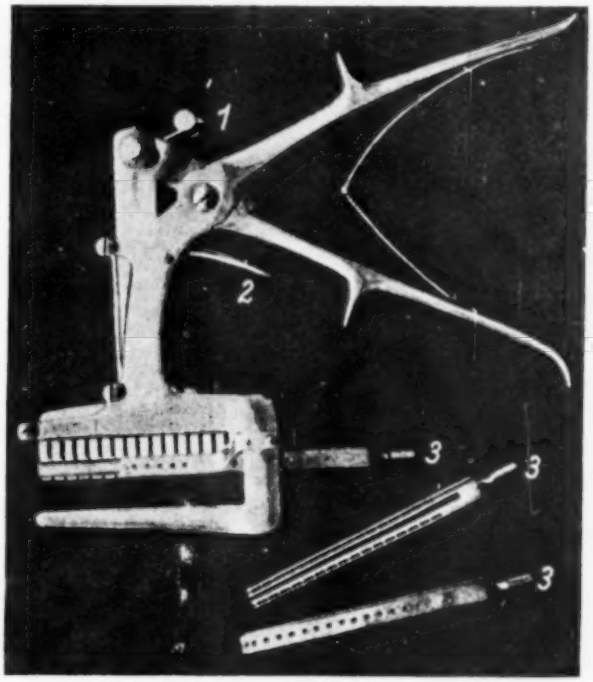


Fig. 2. Intestinal sewing clamp after Neuffer.

the grasped part. Further manual pressure pushes the loaded magazine, seen at the upper branch, into the teeth which are attached to the upper jaw. The clips in the magazine are driven forward into the tissue.

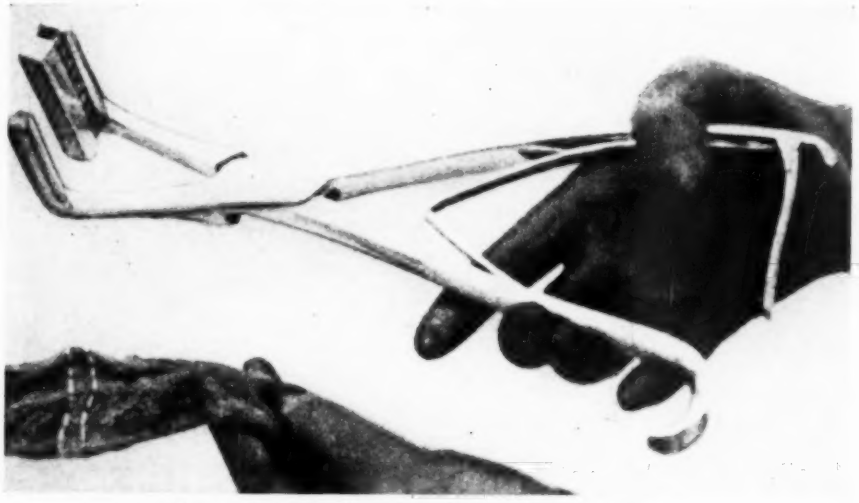


Fig. 3. Intestinal sewing clamp after v. Seemen.

The latest form, which was made at the suggestion of v. Seemen, is shown in Figure 3. It is the simplest and handiest. The

Besides the simplicity of handling this instrument, it has still another advantage. It is built at such angles that parts of the

stomach or intestine, otherwise difficult to be reached, can easily be grasped and sutured.

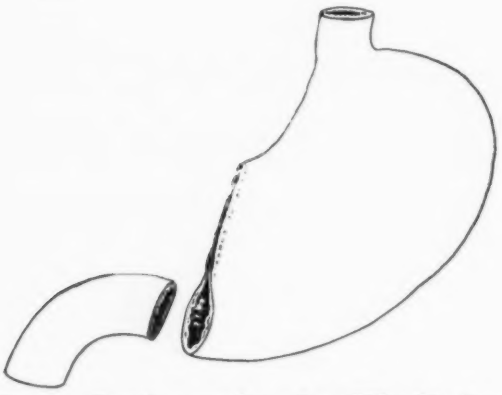


Fig. 4. Gastric resection after Billroth's first method, performed with the intestinal sewing clamp.

It is of importance to know what becomes of the metal hooks pressed into the intestinal wall and covered by the inverting suture. From my own experience with the v. Petz and v. Seemen clamps, there was no patient who afterwards complained of trouble arising from the clips. The clips are either thrown off after three to six weeks, and having fallen into the cavity of the organ, are passed out with the stool, because they are round and do not stick to the mucous membrane; or the clips become encapsulated and remain for a long time—perhaps for life—in place. The hooks made of new-silver seem not to irritate the tissue. A systematic examination was made recently by O. Schurch of Clairmont's clinic in Zurich. Twenty-two patients were examined upon whom the v. Petz clamp had been employed, between one month and three years before. In eleven patients, the clips were still visible. None of these patients were inconvenienced.

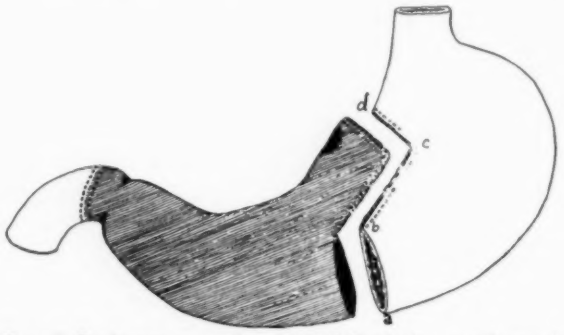


Fig. 6. Staircase resection after Schmieden, performed for ulcers situated higher up at the lesser curvature than usual.

In order to demonstrate the use and advantage of a sewing clamp, I should like to describe some examples of gastric and intestinal surgery:

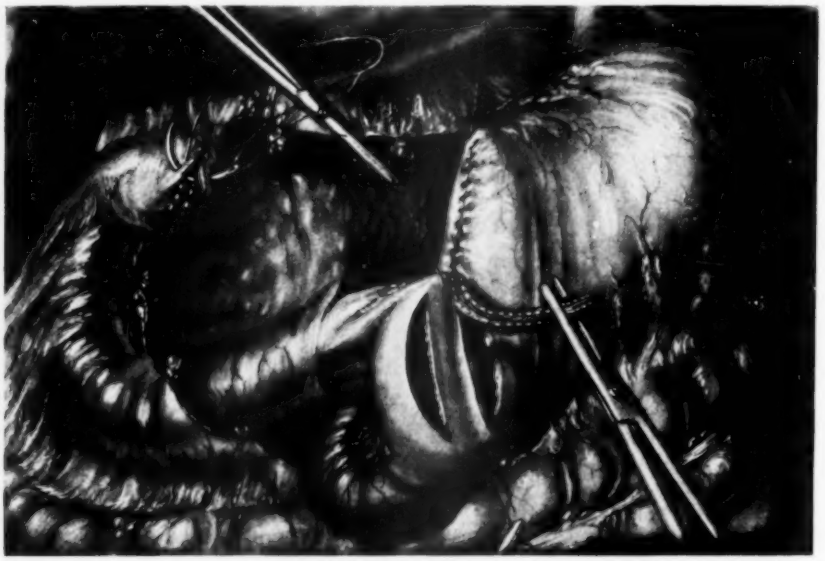


Fig. 5. Billroth's second method (oralis inferior) performed with the intestinal sewing clamp.

Method after Billroth I. Original method. (Fig. 4.) The line of intended section of the stomach is sutured in above de-

scribed way and the organ divided between the two rows of clips. The clips of the proximal part are buried by a Lembert suture with the exception of the lower part. The lower stitches are removed by scissors closely beneath the clips as far as the duodenum is wide. The anastomosis between the stomach and duodenum is done in the usual manner.

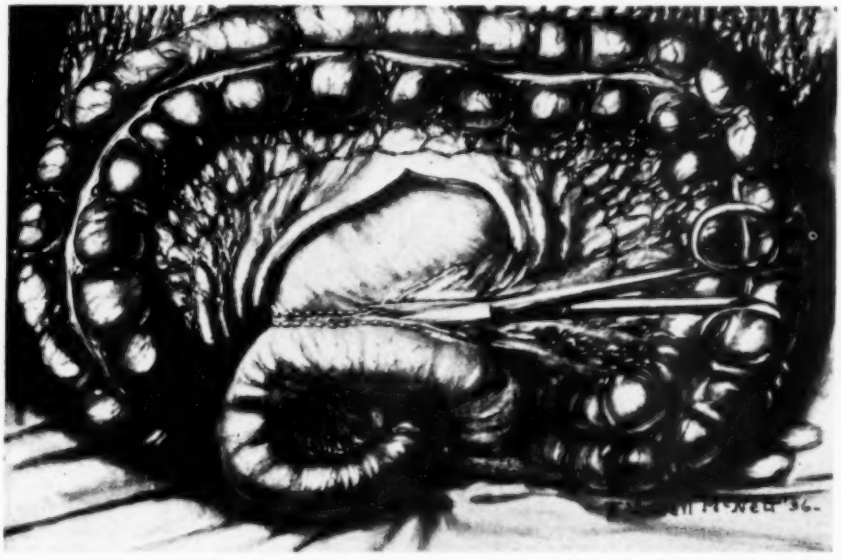


Fig. 7. Removal of a posterior gastroenterostomy with the intestinal sewing clamp.

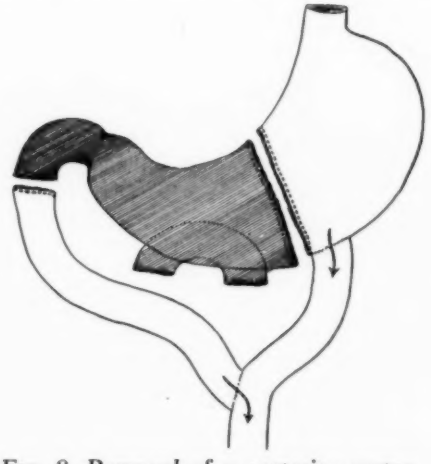


Fig. 8. Removal of a posterior gastroenterostomy together with the corresponding part of the stomach. Y method after Roux.

Method after Billroth II. With this method, the closure of the duodenum is characteristic, and is easily carried out by the employment of the sewing clamp. At the stomach itself, the sewing clamp is of no help when the entire transverse section of the organ is to be used for the anastomosis. But, in the original method, as well as in the rest of the modifications, which all aim to narrow the lumen of the proximal part of the stomach, the employment of the sewing clamp is of great service. One instance might illustrate the situation.

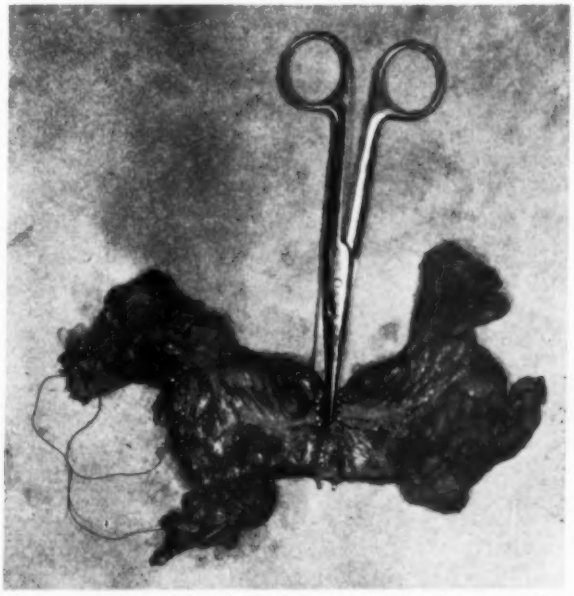


Fig. 9. Separation of the large intestine between the double row of metal clamps and burying them with a Lembert suture.

Billroth II, Oralis Inferior. (Fig. 5.) The clips of the proximal part of the divided stomach are inverted by a Lembert suture with the exception of the lower third. The posterior gastric wall at the lower third is brought in connection with the corresponding jejunal loop by continuous or interrupted posterior suture of the serosa. The clip suture is now removed at the site of the anastomosis, the jejunum at the corresponding site opened, and the anastomosis completed in usual manner.

Especially useful is v. Seemen's sewing clamp in the so-called staircase resection of the stomach after Schmilden. Suppose the seat of the ulcer is higher up than usual at the lesser curvature; a transverse section above the ulcer would leave only a small part of the stomach. In such cases the staircase resection can be resorted to. (Fig. 6.) Between two rubber clamps the stomach is divided from a to b, then the sewing clamp is applied between b and c, and this part first divided before the sewing clamp is again applied between c and d. After partcd has been separated between the stitches, the clips are buried by a Lembert suture and the anastomosis between stomach (ab) and jejunum is carried out as usual.

If a posterior gastroenterostomy must be removed for some reason, usually the afferent loop of the jejunum is too short to allow a side-to-side re-anastomosis of the intestine and an end-to-end re-anastomosis narrows the intestinal lumen. Therefore a technique, such as Figure 7 shows, simplifies the operative procedure. The proximal row of the stitches closes the stomach, the distal row the jejunum. The anastomosis is separated between the two rows and the clips at the stomach and at the jejunum are buried by a Lembert suture. If the anastomosis must be removed together with the corresponding part of the stomach, the Y method, after Roux, in all its variations, is the best in order to avoid the above mentioned difficulties. (Fig. 8.)

In intestinal resections or separations (Fig. 9), the use of the sewing clamp is especially advantageous, not only because of its shortening the operative procedure, but due to the fact that the operative field is kept clean and dry, it is an inestimable advantage in operations on the large intestine. The use of the v. Seemen clamp simplifies considerably the separation of the large intestine deep down in the pelvis, or dividing the gut in the sacral vault in amputations of the rectum by the sacral way.

SUMMARY

1. The employment of sewing clamps in intestinal surgery makes the operative procedure quicker, simpler and more aseptic. The best kind is the sewing clamp of v. Petz and its modifications by Neuffer and v. Seemen.

2. Examples of intestinal surgery are described to illustrate the usefulness of the intestinal sewing clamp.

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

THE MAKING OF A SURGEON*

GATEWOOD, M.D.

Clinical Professor of Surgery, Rush College, University of Chicago; Attending Surgeon, Presbyterian Hospital, Cook County Hospital, Highland Park Hospital

CHICAGO, ILLINOIS

IN considering any topic, definition of terms is imperative that we may all talk the same language. "The making of a surgeon"—out of the fifteen or more definitions of the word "make," I have chosen "to form out of given material," as it is obvious that one does not "create" a surgeon any more than one "compels" him. But just what or who is a surgeon? Etymologically, the word "surgeon," coming from the Greek "cheirourgos," literally means "one who works with his hands," and for many centuries surgery was little more than a mechanical art. Before we attempt a modern definition, first let us—

" . . . delve into the depths of Time
And bring from out the ages that have rolled
A few small fragments of those wrecks sublime
Which human eye may never more behold."

The history of surgery, as suggested by Morris,[1] may readily be divided into: (1) the Heroic Period, embracing all of surgery prior to the development of sound anatomical training; (2) the Anatomic Era, beginning with Vesalius; (3) the Pathologic Period, beginning with John Hunter, and exemplified by Morgagni and by the German school of Virchow and Billroth; and (4) the Physiologic Era. Much of our physiologic information must still be derived from our pathological studies, which in turn are based upon an accurate knowledge of normal anatomy. However, the study of living cells and organisms, and their metabolic processes must influence the development of surgery from now on.

Going back for a moment to the Heroic Period, let us begin with a few scattered fragments of that primitive man of the paleolithic and neolithic age. As Robinson[2] has said so well, even in primitive medicine, as in modern, the patient's faith and the physician's personality were important factors in the cure, and both are still too often the victims of ignorance and suggestion. We have no monuments to that jungle colleague who first controlled hemorrhage by pressure, or to him who sutured wounds by having the edges pinched by the keen nippers of ants while he rapidly severed their bodies. Our primitive civilization goes back a mere six or eight thousand years and what remains to us of this great pre-

* Presented at the First Annual Meeting of the St. Paul Surgical Society, St. Paul, Minnesota, April 9, 1936.

historic era are the fragments which the paleontologists painstakingly unearth with their spades, or the cave-hunters discover in the primitive home of the all-but-forgotten man. These cave discoveries do tell us that the trephining operation was frequently performed 10,000 years ago. The more recent discoveries in Peru prove that the operation was successful by the fact that healing took place. These early Peruvians evidently knew something of asepsis since in embalming they used Peruvian Balm, menthol, tannin and resins, all of which are of an aseptic nature.

FIG. 1. Indian flap operation. Twentieth Century modification of the procedure done several centuries B.C.

Recent discoveries unearthed in India,[3] such as the Rig Veda and Bactrian Document, indicate that the birthplace of civilization was in the far East. At least their records go back to 6000 B.C., and in 4000 B.C. a government is described as of the people, for the people and by the people, with no caste system and numerous guilds. Iron and gold were well wrought and surgical instruments were well designed. Susruta (1000 B.C.) enumerates 121 surgical instruments, including obstetrical forceps. Boiling of water and preparation of patients for operation were described. Hospitals for the care of the sick—and not mere isolation camps such as the earliest Roman hospitals were—sewage systems and other sanitary measures are among the advances of an enlightened India.

FIG. 2. The Italian flap graft method described by Tagliacozzi in 1597.

The Indian operation for building a nose from the forehead was developed during this period. (Fig. 1.) Legend states that custom permitted the disgruntled husband to cut off the nose of his unfaithful wife. Some men, repenting of their haste, desired reconstruction of the noses of their unjustly persecuted spouses and a school of plastic surgery developed.

Recently, while visiting in the mountains of North Carolina, I met a young woman who had unfortunately lost her nose in childhood as the result of a horrible burn. A very excellent reconstruction had been made, taking a flap from her arm. In explaining the operation to me she said, "I was fortunate enough to go to Johns Hopkins just after this method was discovered." She would have been more nearly correct had she said "re-discovered," for here is a picture of the operation as performed by Tagliocozzi in 1597. (Fig. 2.)

Buddhism, beginning about 600 B.C., marked the deterioration of medicine in

India, and Aryan civilization migrated to Arabia, Persia and along the shores of the Mediterranean, especially to Egypt and the Islands of Asia Minor.

Egyptian medicine has come down to us largely through the papyruses (2000—1000 B.C.), such as the Ebers and the Smith (1600 B.C.—first rubrics.) These furnish an excellent idea of the medical therapy of the early Egyptians. They also show the treatment of fractures with splints, a method for reducing shoulder dislocations, the Indian operation for amputated noses, etc. Embalming processes taught much anatomy and left behind evidences of disease which we can interpret today. This embalming process varied with the wealth of the deceased (or his relatives). The poor were pickled in some preservative, probably brine, for seventy days and then wrapped in bandages and immersed in some resinous gum. The wealthy like Rameses II and III, were so well preserved that their features were recognizable when they were recently unwrapped. These mummies tell us clearly that the early Egyptians had gall-stones, kidney-stones, pneumonia and various other pathological lesions. Even bacilli can still be stained in some of their abscesses. Votive offerings cast in the form of the supposedly offending organ, give mute testimony of the ancients' knowledge of anatomy.

While Egypt was carefully preserving the traditions of an already aged civilization, a new civilization was developing across the Mediterranean. Homer revered Egypt as the original source of wisdom and stated in the Odyssey about 1000 B.C., that "in Egypt men are more skilled in medicine than any of human kind." (Book IV, 227.)

Greek culture reached its prime in the age of Pericles following the battle of Salamis (480 B.C.). This age produced such master minds as the world has probably never equalled in the same space of time: Herodotus the historian, Sophocles the tragedian, Socrates, Plato and Aristotle—the greatest philosopher of all time—and Hippocrates, the father of medicine. (Fig. 3.) Born on the Isle of Cos, off the coast of Greece, in 460 B.C., Hippocrates travelled much and observed widely. He may not

Fig. 3. Hippocrates as shown in the surgical works of Ambroise Paré.

have written much that was new, but his keen judgment enabled him to accumulate an enormous amount of medical information, most of which was preserved to posterity. His treatises on fractures and dislocations contain much which is good today. His aphorisms reveal his mastery of conciseness. The best known of these is; "Life is short, and the art long; the occasion fleeting; experience fallacious and judgment difficult. The physician must not only be prepared to do what is right himself, but also to make the patient, the attendants and externals coöperate." That satirist, Petrarch, in discussing physicians and their ignorance, said; "Life in itself is short enough, but the physicians with their art know to their amusement how to make it still shorter."

But let us turn for a moment from the culture of Greece to that fearless, capricious, conceited Alexander of Macedonia. Seeking more worlds to conquer, he invaded Egypt, founded Alexandria "on an island in the much-dashing sea, in front of Egypt." According to Robinson, he never saw the city again although his body was brought back nine years later for burial. Following the collapse of his empire, in the division of the spoils, to

the lot of Ptolemy Soter, the shrewdest of his generals, fell Egypt. Then followed the reign of the Ptolemies. (We are apt

FIG. 4. Galen (130–200 A.D.) the father of experimental medicine.

to forget that they were Macedonians who closely intermarried as a rule and that Anthony's Cleopatra had not a single drop of Egyptian blood in her royal veins!)

Ptolemy Philadelphus (the second in the dynasty) was noted for his collection of books and women. The University of Alexandria was established in the Fourth Century B.C. The library of Alexandria became the magnet of attraction for scholars of Greece and Asia Minor, and here in mathematics and astronomy, in physics and mechanics, Alexandria outdistanced the world. The fame of the school of medicine spread far and wide. Herophilus distinguished arteries from veins and described the pulse in health and disease. He named the duodenum and accurately described the female reproductive organs. He was probably the first teacher publicly to dissect. The torcular Herophili serves today to remind the medical student of his anatomical knowledge.

Archimedes, the mathematician who declared, "Give me whereon to stand and I will move the world," invented the endless screw and the tripaston which were adopted to reduce dislocations. Ammonius, using the screw of Archimedes to produce the crushing force, invented the lithotrite because he found stones too big to remove.

The death of Archimedes is symbolic of the conquest of Greece by Rome. Rome gave to the world law, roads, aqueducts and history, but not medicine. Most medical men in Roman history were itinerant Greeks, the greatest of whom was Galen (130–200 A.D.) (Fig. 4.) His prolific writings, which brought to the Latin language innumerable Greek words for which there were no Latin synonyms, were the holy writ of medicine for the next 1200 years. (Medically speaking, "the Greeks had a word for it" is far more fact than fancy.) Although Galen was an egotistical bigot, he was possessed of indefatigable energy and his numerous experiments made him justly referred to as the father of experimental medicine. Even before his death, Rome, softened by luxury and lust, was beginning to decay, and within the next two centuries that vast empire, extending from Great Britain to India, became the crumbled wreck of the despised Barbarians of the north. What fragments of learning remained in Europe were locked in the monasteries of the monks and medicine reverted to the Dark Ages. Through this long night during which European culture almost vanished, the traditions of surgery were carefully preserved by the Arabs. The teachings of Hippocrates and Galen were painstakingly translated and transcribed so that copies of their works were widely read. However, the Koran prohibited dissection and the Arabian mind was much more concerned with the compilation of knowledge than in initiating inquiry. Avicenna's (980–1037 A.D.) Canon, containing about a million words, is the summation of Graeco-Arabian medicine, but it adds little to the advancement of surgery.

After the darkness of succeeding centuries, the first gleam of dawn was seen in the University of Bologna. Mundinus

lectured there between 1315 and 1325, and publicly dissected although still under the spell of the Galenic traditions. Two centuries later, Andreas Vesalius (born in 1514 and dying in 1564, the year of Shakespeare's birth), the greatest of anatomists, swept away the out-worn "analogical" anatomy with the production of his treatise "De Corporis Humani Fabrica Libri Septem," published when he was 29. These books marked the beginning of artistic illustrations as well as the Anatomical Era. (Figs. 5 and 6.) While the plates have been attributed to various renowned artists of the Renaissance, there is now little question but that the majority are the work of John Stephen de Calcar, a pupil of Titian. In a warm climate and without preservatives, the task of Vesalius and his artists is little short of miraculous. Compare the accuracy of the plates of Vesalius with those of contemporaries still under the influence of Galenic teachings.

Fig. 5. Drawing from Vesalius' anatomy showing combination of accurate delineation and artistic skill.

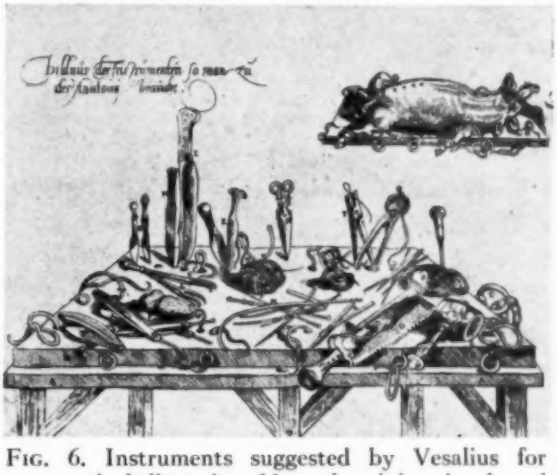


Fig. 6. Instruments suggested by Vesalius for antomical dissection. Note the vivisection board with its almost modern arrangement. (Figs. 5 and 6 are taken from an original text belonging to the Rush Library. The yellowness of the paper accounts for the lack of clearness of the cuts.)

(Fig. 7.) Note the multiple lobes of the liver such as are found in the dog and the lower animals in this illustration published in the Fifteenth Century. Vesalius' work was not limited to anatomical demonstrations, but includes treatment of fractures and even a chapter on vivisection. Notice how the victim, a pig, is restrained in the modern manner on a dissecting board. (Fig. 6.) Despite the abuse and criticism of such men as his old teacher, Sylvius, whose aqueduct has immortalized him to medical students, his work was widely accepted and surgery entered the anatomical era.

Methods depending upon clinical and experimental observations had found as yet little place in scientific surgery. In France, however, was born in 1510, a man whose influence on the future of surgery has been far greater than that of anyone since Galen. This man was Ambroise Paré (Fig. 8), often referred to as the father of surgery and certainly the *père* of French surgery. He was endowed with one of the greatest original minds our art has known, fearless, independent and alert. The excellent translation by

Packard of "The Apology and Treatise by Ambroise Paré of Laval, Councillor and Surgeon to the King" should be in the

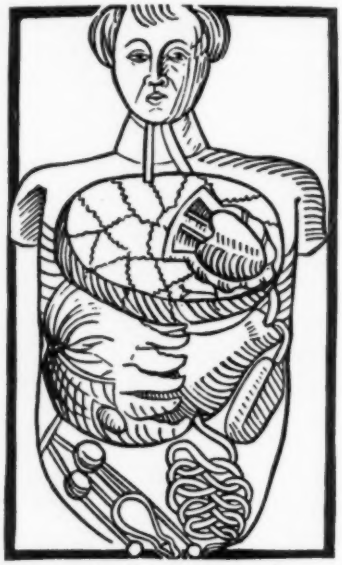


Fig. 7. Anatomical drawing—still under the influence of Galenic teachings. Contemporary with Vesalius' drawings.

library of every surgeon. His proof of the superiority of the ligature over the cautery as the ideal method of controlling hemorrhage, reads like a fascinating novel. In the Apology, he sets forth the attack of his adversaries and then responds thus, "Oh, what beautiful words! for an aged man, who calls himself a wise doctor. He does not remember that his white beard admonisheth him not to say anything unworthy of his years, and that he should put off and drive out from him all envy and rancor conceived against his neighbor. But, now I wish to prove to him by *authority*, *reason*, and *experience*, (the italics are mine) that the said veins and arteries should be tied." He succinctly quotes from "that grand man Hippocrates," from Galen, Gui de Chauliac, Vesalius and others to prove ample authority for the use of the ligature. Then follows an empirical argument refuting the allegations of his opponents, and finally under the heading "Experience" he cites case after case, giving the exact address of the patients, the names of the barber-surgeons assisting, as well as those of other witnesses. I cannot resist citing one of these histories:

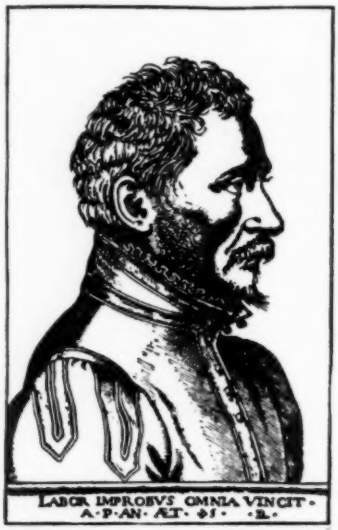


Fig. 8. Ambroise Paré. The father of modern surgery.

"In the year 1583 (Paré was then 73, and still in active practice) the tenth day of December, Toussaint Posson, native of Roinville, at present dwelling at Beauvais near Dourdan, having his leg all ulcerated, and all the bones carious and rotten, besought me that for the honor of God I would amputate his leg, because of the great pain which he could no longer bear. After being prepared, I had his leg amputated four fingers below the rotula (patella) of the knee, by Daniel Poullet, one of my servitors, to teach him and enbolden him to do such work, where he tied very dexterously the vessels in order to staunch the blood, without the application of hot irons, and in the presence of Jacques Guillemeau, surgeon in ordinary to the king, and Jean Charbonnel, master barber-surgeon in Paris. During his cure he was seen and visited by Messieurs Laffilé and Courtin, doctors regent in the Faculty of Medicine of Paris.

"The said operation was performed in the house of Jean Gohel, innkeeper, dwelling at the sign of the White Horse in the Grêve." (When I cited this history to my house staff as an illustration of the impor-

tance of a detailed history, they replied that it also demonstrated the importance of permitting the younger men to operate.)

The revival of dissection marked the decline of philosophical speculation in surgery and the beginning of scientific investigation. The unfortunate Michael Servitus, who was burned with his works as a heretic at Geneva because he incurred the displeasure of Calvin, undoubtedly recognized the pulmonary circulation, as proved by the two extant copies of his works missed by the fagots of his bigoted persecutor. However, it remained for an Englishman, a pupil of Fabricus at the same University of Padua where Vesalius had been asked to lecture at the age of 21, to describe the circulation. William Harvey began his epoch-making experiments in the year of Shakespeare's death. He writes, "When I first gave my mind to vivisections, as a means of discovering the motions and uses of the heart, and sought to discover these from actual inspection and not from the writings of others, I found the task so truly arduous, so full of difficulties, that I was almost tempted to think with Frascatorius, that the motion of the heart was only to be comprehended by God . . . At length and by using greater and daily diligence, having frequent recourse to vivisections, employing a variety of animals for the purpose, and collecting numerous observations, I thought that I had attained to the truth."[4]

With the value of experiment established in science, research in surgery was inevitable and the next century saw the development of deductive reasoning based upon clinical and experimental data. Pathological anatomy replaced traditional speculation in the etiology of disease. Bearing the standard of surgical pathology, John Hunter (1728–1793) found surgery a mechanical art and left it an experimental science. One of his colleagues said of him, "He alone made us gentlemen." His rebuff to one who accused him of ignorance of the classics is famous; "Jesse Foote accuses me of not understanding the dead languages: but I could teach him that on the dead body which he never knew in any language, dead or living." Still preserved in the museum which he founded, I have seen his experiments on growth of bone by which he showed just as conclusively that longitudinal growth occurs solely at the ends (epiphyses) as I[5] could demonstrate it with the *x*-ray and all modern laboratory facilities. (Fig. 9.)

With the dawn of the 19th century came a new epoch in the progress of surgery. The pathologists, aided by improved microscopic eyes, resolved malignancies into separate entities, and the physiologists, by scientific experiment, began to unravel the mysteries of the function of organs in health and disease. One cannot help feeling proud of that young American army surgeon who seized his opportunity to demonstrate the action of the stomach and its juices. The pertinacity of William Beaumont in dealing with his recalcitrant subject in a remote post in the wilderness should be an inspiration to anyone engrossed in a modern laboratory problem.

Hemorrhage, pain and infection, the three great evils which had always embittered the practice of surgery and checked its progress, were in a brief quarter of a century (1846–1873) robbed of their terrors. The discovery of anaesthesia, in 1846, stimulated the development of meticulous surgical technic but infection still remained the *bête noir* of surgeon and patient alike. It was in 1856 that the great French chemist Pasteur[6] began his experiments which finally demonstrated that micro-organisms were the cause of putrefaction, and that specific germs produced specific disease. Though not educated in a medical school, he became one of Medicine's heroic figures. It was he who said: "Without theory, practice is but routine born of habit. Theory alone is able to call into being and develop the spirit of invention." It would seem that many so-called research men are unacquainted with this aphorism.

In 1827, there was born in England a young Quaker of whom Lord Moynihan has said; "If a man's services to humanity are the standard by which we measure his value, then Lister may be counted as perhaps the greatest man the world has produced." The son of a scientist who contributed much to microscopy, although always an active tradesman, young Lister graduated in London and became house officer on Syme's busy service in Glasgow. There he read of Pasteur's work and realized its significance in the prevention of inflammation, a problem in which he had become vitally interested on account of the frequency of its disastrous consequences. In 1867, he published his first account of the antiseptic method. As in the case of most major prophets, his own countrymen were slow in recognizing the importance of his work, but his method was adopted almost immediately in Germany and only a little later in America.

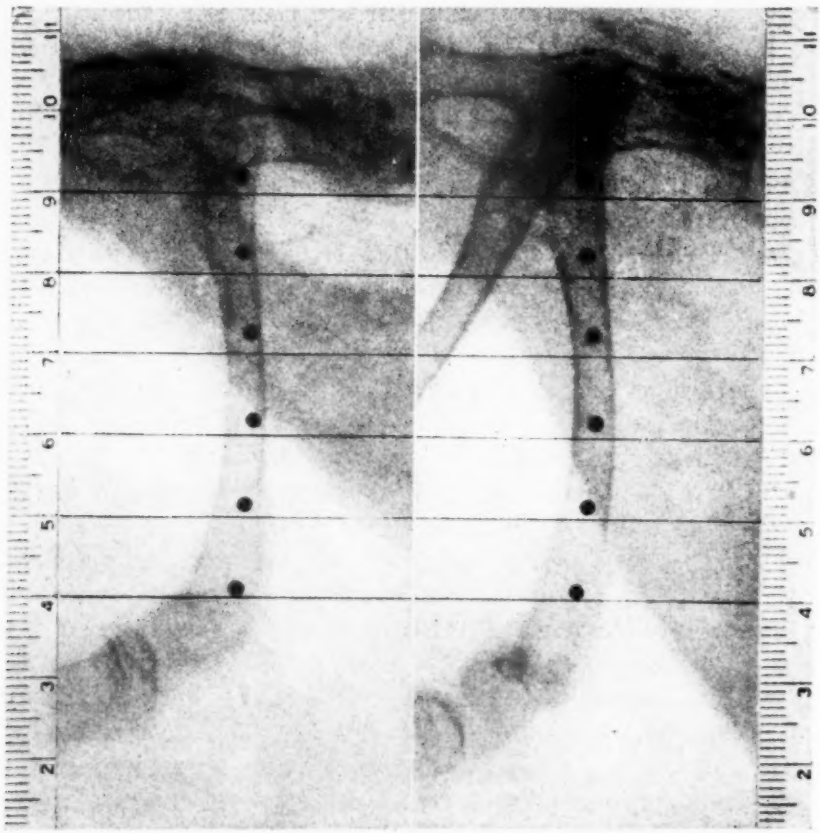


FIG. 9. Roentgenograms of rabbit femur three and forty-four days after introduction of shot, showing that the longitudinal growth is exclusively in the epipysis. (GATEWOOD and MULLEN. Archives of Surgery, August 1927.)

The achievements of clinical research have been gigantic since Lister's day. Much remains to be done, but the way is clearly marked. The future of surgery depends upon clinical and physiological research. An organism may be perfect anatomically but it needs the elusive spark of life to give it being. The mechanical era, based upon accurate anatomical and deadhouse information, is being steadily augmented by the study of the changes occurring in living tissues and their interreactions in the living organism. A few years ago, the Nobel Price was awarded to Warburg who demonstrated that metabolic processes are different in normal and malignant cells. Physiological changes do not necessarily parallel those seen in the pathological laboratory. Billroth, that master technician who made the first successful stomach resection for cancer, warned that surgical interference should lead only toward a physiologic end. Too often

anastomoses of the digestive tract are more closely related to the art of the plumber than to the science of physiologic research. That the ablation of an organ produces no immediate demonstrable change in the individual is no proof that it has no function. The parathyroids made their appearance on the anatomic horizon only about fifty years ago, and would still be anatomic curiosities were it not for the experimental observations of the physiologists. At least fourteen different actions have been attributed to extracts of the hypophysis, some direct, and some indirect by stimulation of other glands of internal secretion. Not so many years ago, Dr. Samuel Gross, then President of the American Surgical Society, said, "All avenues of approach to surgery have been investigated." Nothing remained except improvement in technique. What would have happened had that young Canadian orthopedic surgeon, Banting, believed this? The discoverer of insulin would still be plain surgeon F. G. Banting, and not Sir Frederick Grant Banting, the conservitor of more lives than any general in the history of this troubled world.

In this brief survey of the development of surgery, it seems to me that the qualifications for the ideal surgeon become self-evident. He must be a physician possessed of the high ideals of a Hippocrates, the anatomical knowledge of a Vesalius, the alertness and fearlessness of a Paré, the intuitions and curiosity of a Hunter, the imagination of a Pasteur, and the industry and honesty of a Lister. To these must be added that good judgment which marks the difference between technical efficiency and true ability. Well grounded in the fundamentals of anatomy, pathology and physiology, a master of method and technique, he must be further trained in the art as well as the science of *Medicine*. The advice of Thomas Vicary[7] is as good today as it was in 1577, "Every chirurgion ought to be learned and that he know his principles, not only in chirurgie but also in phisicke that he may better defend his chirurgie."

Equipped with a clear conception of our task, the difficulties encountered in the making of a surgeon are in no way lessened although the problems is somewhat simplified. The finished product must depend both upon the ability of the *maker* and the *material* at his disposal. Recent German literature emphasizes the physical requirements necessary to the capable surgeon and a strong physique is undoubtedly an asset to the successful one. There is probably no profession which makes greater demands upon the physical reserve of the individual than that of the surgeon. The training of a surgeon should include methods of conserving energy. The necessity for vacations and the importance of a real hobby should not be forgotten.

The essential ingredients, however, to quote Royster,[8] are the mental, the moral and the mechanical. "The first has to do with knowledge, the second with judgment and the third skill . . . Happy indeed is he—and rare in the extreme—who may possess them all in equal degree." Medical educators everywhere are endeavoring to devise more intelligent methods for the selection of the students who are to become our future doctors. Occasional outstanding blunders should not be held against the methods. The results on the whole need no vindication. The medical student today is often more alert than his teacher. Freed from the fetters of outworn dogmata, it is our duty to keep him free.

"How happy is he, born and taught,
That serveth not another's will,
Whose armour is his honest thought,
And simple truth his utmost skill."

With the improved caliber of doctors furnished by our medical schools, the great responsibility for the selection of the proper material for further development rests squarely upon our shoulders. The process in the past has been largely a matter of self-selection with a certain amount of survival of the fittest. Genius will not be downed and outstanding figures have developed just as they have in music, art and literature. However, this trial-and-error method is often hard both on the

individual and on the community, and much good may be accomplished by more careful selection of the young men who are to devote their lives to surgery.

Selection too often depends upon grade points. Of equal importance are those intangibles which go to make up personality. As Kanavel[9] says, they may have no ponderable value but they mark the difference between technical efficiency and true greatness. I refer to honesty, both with one's self and with one's fellow-man, love of the search for knowledge, culture, imagination and common-sense. Inherent in a large degree, these qualities can be improved by thought and study. I know of no adequate test for determining these intangibles. Familial and racial backgrounds are of some importance, as are breeding and a broad general education, but intimate association with candidates over an extended period of time will do more to prevent disastrous mistakes than any set questionaire.

And now, a few words about the *maker*. It must be kept in mind that the teaching of surgery cannot be divided artificially into periods or stages. It is a continuous educational process from admission to the medical school to retirement from practice. Many of the faults in the training of surgeons begin at the bottom and continue to the very top. Good habits inculcated during the formative period of training will persist throughout the surgeon's life. It probably makes no vital difference whether that training be by apprenticeship under a master, or by the pyramid system of surgical residencies provided in most of our teaching hospitals. Both methods have their obvious advantages and drawbacks. The first is more apt to make a surgeon with astigmatic vision, albeit he bears the stamp of a true master. On the other hand, large surgical clinics may become soulless factories in which the student's initiative and imagination are stunted and he becomes a mere cog, learning a mechanistic surgery without ideals or inspiration.

Technical excellency, the marvel of the undergraduate and the envy of the young surgeon, is of only relative importance and is the natural result of adequate training. It should always be subjacent to surgical judgment. It is vastly more important to stimulate an inquiring mind to reason than to train the willing hand to manual dexterity. Since modern surgery has become the work of a team in which each plays an exacting part, it would seem that the ideal training will consist of a varied instruction in an institution directed and led by a master such as a Halstead or a Billroth.

Finally, in a simple, somewhat sketchy way, I have attempted to outline the problem of the making of a surgeon. I have tried to indicate principles, and purposely have omitted boring details. There are two variables involved, the maker and his material. To each must be given somewhat intangible values. The result will approach the ideal only when more careful attention is given to the selection of the material and to the caliber of the maker, including you and me. For, whether we wish it or not, whether we like it or not, and whether we are a part of a teaching clinic or not, we, you and I, must inevitably play a part in the development of the next generation of surgeons. Let us keep faith with our traditions and make our contribution worthwhile.

REFERENCES

1. Morris, Robert T. Fifty Years a Surgeon. E. P. Dutton & Co., Inc., N. Y., 1935, p. 149.
2. Robinson, Victor. The Story of Medicine. New York, 1931, Albert and Charles Boni.
3. Muthu, D. Chowry. The Antiquity of Hindu Medicine and Civilization. 3rd Edition. New York, 1931, Paul B. Hoeber, Inc.
4. Packard, Francis R. Life and Times of Ambroise Paré (1510–1590). New York, 1921, Paul B. Hoeber, Inc.
5. Gatewood and Mullen, Bernard P. Experimental Observations on the Growth of Long Bones. *Arch. Surg.*, 15: 215, 1927.
6. Vallery-Radot, René. The Life of Pasteur. New York, 1920, Doubleday, Page & Co.
7. Turner, G. G., Hurst, A. and Ross, J. P. Discussion on Training of a Surgeon. *Proc. of Roy. Soc. of Med.*, 28: 1239–1250 (July) 1935.
8. Royster, H. A. Surgical Vision. *J. A. M. A.*, 99: 93 (July) 1932.
9. Kanavel, Allan B. Intangibles in Surgery. *Surg., Gynec. & Obst.*, 56: 394, 1933.

BOOK REVIEWS

CLINICAL ELECTROCARDIOGRAPHY. By Sir Thomas Lewis, M.D. Sixth Edition. England, 1937. Shaw and Sons Ltd. Price 8s. 6d. net.

This well known book, which has gone through five previous editions, is intended to serve as an introduction to students of electrocardiography and as a guide to practitioners and hospital physicians in understanding curves that may be taken by others from patients in their charge.

This small book (128 pages) covers: The Electrocardiographic Method, The Physiological Electrocardiogram, Rhythmic But Anomalous Electrocardiograms, Auriculo-Ventricular Heart-Block, Premature Contractions or Extrasystoles, Simple Paroxymal Tachycardia, Auricular Flutter, Auricular Fibrillation, Sinus Disturbances and Alternation, and Special Conditions.

There is a good Index and 109 Figures are included.

A TEXTBOOK OF HISTOLOGY. By Harvey Ernest Jordan, A.M., PH.D. Seventh Edition. New York, 1937. D. Appleton-Century Company. Price $7.50.

This book was first offered physicians and medical students in 1916. It is not surprising that it is now offered in a seventh edition. It had been accepted as a fixed standard in its line. And because it has fulfilled its purpose it is but natural that it has had a wide distribution.

This new edition adheres to the general principles along which the earlier editions were planned. Occasional brief functional interpretations and physicochemical explanations are given, but these are not allowed to overshadow structural details. Thirty-nine figures have been replaced by new and improved illustrations. Of the total of 610 illustrations, 19 are from the collection of Professor Irving Hardesty of Tulane University. Brief additions to the text have been made covering newly revealed functional features of periosteum, ovaries, suprarenals, thymus and pineal gland.

DISEASES OF THE HEART. By Sir Thomas Lewis. New Second Edition. Completely revised. New York, 1937. The Macmillan Company. Price $3.50.

If we were asked to name one book for a physician or medical student on the subject of heart disease for use prior to referring to larger works, we would instantly direct the questioner to Sir Thomas Lewis' "Diseases of the Heart."

He says: "The impulse to write a work of reference has not stirred me, but I have had the desire to place at the disposal of students and medical practitioners the outline of my clinical teaching on diseases of the heart, as this has developed in my talks to my own hospital students." The author being internationally known as one of the outstanding, if not the outstanding authority in this field, a small book, (297 pages) written to the point, and covering all phases of diseases of the heart in such a manner that even a third or fourth year medical student can "get" the fine points, we name this book as one that should be owned by every medical student and medical practitioner. This should not be a burden, for the cost is most moderate.

The first edition appeared in 1933. This, the second edition, is welcome, and no doubt in the future many other editions will become necessary.

Surgeons who wish a book on this subject need look no further afield.

PRACTICAL TALKS ON KIDNEY DISEASE. By Edward Weiss, M.D. Springfield, Ill., 1937. Charles C. Thomas. Price $3.00.

This is an interesting and very readable book, and although the author tells us it is not intended as a monograph or a textbook, physicians will find they can read this book with profit.

In the preface the author states: "If a physician has had a serious illness he is apt to develop a special interest in the disorder from which he has suffered. Many specialists are made this way. Ever since an attack of glomerulonephritis during my interneship I have been especially interested in kidney disease." Hence this book of 176 pages covering General Considerations, Disturbances of Renal Function, Signs and Symptoms of Kidney Disease, Glomerulonephritis, Nephrosis, Pregnancy and

Kidney Disease, and Nephrosclerosis. There is an Index.

PRACTICAL ENDOCRINOLOGY. SYMPTOMS AND TREATMENT. By Max A. Goldzieher, M.D. Second Edition. Illustrated. New York, 1937. D. Appleton-Century Co., Inc. Price $5.00.

From month to month something new is added to this broad subject. Few physicians know more concerning this topic than they read in the "medical pamphlets" sent them by pharmaceutical houses. Therefore, this book (344 pages) is welcome. And one can well understand why it comes to us in a Second Edition. Physicians in every field of medicine must have more than a superficial understanding of this subject and they would do well to read carefully Dr. Goldzieher's text before going on to larger, more involved textbooks.

The book thoroughly covers the subject. We list only a few chapter headings: Morphology of the Endocrine Glands, Physiology of the Endocrine Glands, Examination of the Endocrine Patient, Disturbances of Development, Growth, Nutrition, Metabolism, Fat Metabolism, Protein Metabolism, Calcium Metabolism, Salt and Water Metabolism, and Iron Metabolism; Disturbances of Heat Regulation, Disturbances of Circulation, of the Digestive Tract, of Locomotion, of Sex; Disturbances of the Endocrine System in Pregnancy; Therapy; and there are chapters on the treatment of the many and various endocrine disturbances.

There is a Supplement.

DISEASES OF THE NERVOUS SYSTEM IN INFANCY, CHILDHOOD, AND ADOLESCENCE. By Frank R. Ford, M.D. Springfield, Ill., 1937. Charles C. Thomas. Price $8.50.

This is a solid work of 953 pages. The reviewer finds it an excellent book. Also, it is a book that is timely and fills a gap in the literature. It should enjoy a wide distribution among neurologists, pediatricians and physicians who come in contact with humans up to and through the age of adolescence.

The author in his Preface says; "The diseases of the nervous system of infancy and childhood constitute a very difficult field which belongs to both the neurologist and the pediatrician, but which has not been cultivated intensively by either. The neurology of childhood is still in an early stage of development as contrasted with that of adult life, and even the common neurological disorders of this period are, to a great extent, obscure. I have attempted, therefore, to bring together and to analyse all available information about these conditions in the hope that I may be able to clarify the situation to some extent."

The reviewer will not pause to select some minor point, controversial in type, and resort to a one-sided debate. It would be in poor taste. Throughout the book the author impresses one as possessing a deep knowledge of his subject, which he offers the reader in a style that is graceful and ever has the earmark of being scientifically sound and true. Here and there throughout the text are bibliographies. The index is ample.

To anyone interested in this subject this book will become the standard. Senior medical students will find it an excellent book to own, and to consult long after their graduation.

Rose & Carless' MANUAL OF SURGERY. American (Fifteenth) Edition. Edited by William T. Coughlin, M.D., F.A.C.S. From the Fifteenth English Edition by Cecil P. G. Wakeley and John B. Hunter. Baltimore, 1937. William Wood and Company. Price $9.00.

The first edition of this work first appeared about forty years ago, and since then it has been issued in fourteen editions. The fifteenth edition, edited by William T. Coughlin, is now offered the profession.

In every sense of the word this is a textbook of surgery (1536 pages; index, 50 pages; 900 illustrations), and being such one need not go into detail regarding the contents. Inasmuch as this monumental work has been rewritten and brought up to date every few years, suffice it to say that this edition contains the latest in surgical thought and procedure, briefly written, presented in an orderly manner, and detailing the facts of the basic groundwork of the science and art of surgery as well as those of its practice.

This, the fifteenth edition, has new chapters added, new illustrations, and much of the former text has been rewritten.

Not a book to attempt to read as one would a monograph, but a good book to have in one's library to consult when necessary. A book appearing in a fifteenth edition needs no bally-

hoo. It has earned for itself a permanent place in the surgical literature of the day.

A Brief Outline of Modern Treatment of Fractures. By H. Waldo Spiers, A.B., M.D. Second Edition. Baltimore, 1937. William Wood & Company. Price $2.00.

This short book (137 pages; 109 illustrations), now offered in a second edition (first edition, 1935) should have a wide appeal to the medical student, the general practitioner, and the surgical interne. The style is direct and the author wastes no words.

The book was written to "outline the modern treatment of fractures," and it "aims to illustrate the fundamentals in bone surgery and to touch only the high spots in their practical application."

The author points out the pitfalls and dangers of fracture surgery, and illustrates some of the more recent "tried and true" methods of treatment.

In the second edition the chapter on "Fractures and Dislocations of the Hip" has been changed to include the Watson-Jones approach. The chapter on "Fractures of the Ankle and Foot" has been extended to include a description of arch support for injured lower extremity. There has been added a short chapter with illustrations on "Fractures of the Mandible and Maxillae."

There is an Index.

The Human Body. By Logan Clendening, M.D. New York, 1937. Alfred A. Knopf. Price $3.75.

This is the sort of book one sees reviewed in the book section of the New York Times or the New York Herald-Tribune. But it came to us for review and we took it home and opened it with an idea we would glance through it, and then bury it on a library shelf, when as a matter of fact we read over 200 of its 443 pages at one sitting and no doubt would have read the rest had we not been called elsewhere. As a result, this book, so full of common sense and cold facts, offered us in a palatable literary form, now in its fourth edition (it has been corrected, enlarged and rewritten) is recommended by us to all physicians for their enjoyment and perusal.

We do not know Dr. Clendening. We have never seen him. But we have read his other works and many of this newspaper syndicated articles, and we picture him as a large man, an engaging conversationalist, a fellow fond of good foods and good books, and one who is not averse to a cocktail when work is over. The textbook stuff you possibly might skip but you will not skip the parts in which he talks informally and airs his opinions and philosophy. You'll read those parts and may disagree in toto, but we feel safe in saying you'll chuckle every few lines. It's gorgeous.

There's nothing to be gained in going into detail regarding the contents. Dr. Clendening has written a book for lay consumption. It has had a large sale. Here it is again offered in a new edition. We throw out the suggestion that a physician can read it without his time being wasted. At least, many physicians might study it with an idea of learning the art of writing on a scientific topic and getting the message over without putting the reader to sleep every few minutes. It beats most best-seller novels.

Operative Obstetrics. A Guide to the Difficulties and Complications of Obstetric Practice. By J. M. Munro Kerr, LL.D., M.D., F.C.O.G. Fourth Edition. With the assistance of Donald McIntyre, M.D., F.C.O.G. and D. Fyfe Anderson, M.D. With 338 illustrations in the text. Baltimore, 1937. William Wood and Company.

When you say a book is an old and tried standard, and has earned the approval of teachers and specialists in a given field, you have said sufficient for anyone in need of such a work to purchase a copy. And this holds true of this volume. There are other good books on operative obstetrics but there are none better than the one under discussion. In every sense of the term it is a textbook, and being a textbook it covers its field in detail.

However, it has another delightful feature; the author knows how to write on a scientific topic and escape dryness and pedagogic dullness.

Naturally, this work is of interest mainly to those engaged in the art and science of midwifery, but any physician or medical student can refer to this volume with the knowledge he will read the last word in scientific obstetrics from an operative standpoint.

The book is 847 pages long, the illustrations are good, and there is a complete Index.

PRACTICAL CLINICAL GYNECOLOGY

HENRY C. FALK, M.D., F.A.C.S.

EIGHTH INSTALLMENT

CONTENTS

[*This Issue*]

Chapter		Vol. xxxviii Page
xiii.	Prolapse —Cystocele —Lacerated Perineum—Rectocele	412

[*Previously Issued*]

		Vol. xxxv
i.	Examination of the Patient	153
ii.	Preoperative Hospital Routine; Preparation of the Patient; Postoperative Hospital Routine	172
iii.	Abortion	187
iv.	Gonorrheal Infection of the Tubes	629
		Vol. xxxvi
v.	Fibroids	559
vi.	Retrodisplacements of the Uterus	750
vii.	Menstruation and Its Disorders	766
		Vol. xxxvii
viii.	Sterility	143
ix.	Ovary	351
x.	Endometriosis	363
xi.	Carcinoma	371
xii.	Ectopic Pregnancy	549

Chapter XIII

PROLAPSE—CYSTOCELE—LACERATED PERINEUM—RECTOCELE

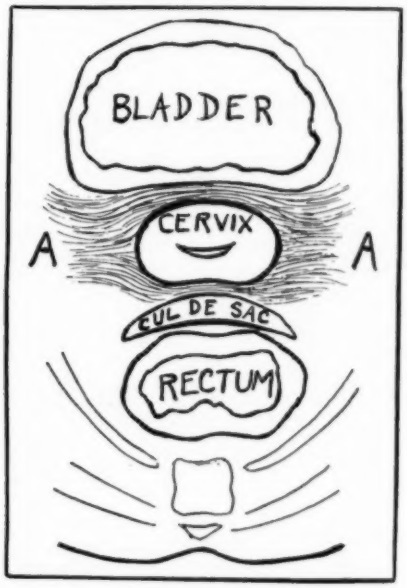
In using the term prolapse it must be clearly understood that it refers only to a descent of the uterus itself. An hypertrophied cervix (congenital or acquired), presenting itself at th eintroitus, or a cystocele, i.e., bulging of the anterior vaginal wall, are both frequently incorrectly called prolapse. These, as well as other conditions, may complicate prolapse and can be corrected at the same operation. It is therefore necessary to have all these conditions separately noted both for the records and as an aid in selecting the type of operation to be performed.

ETIOLOGY

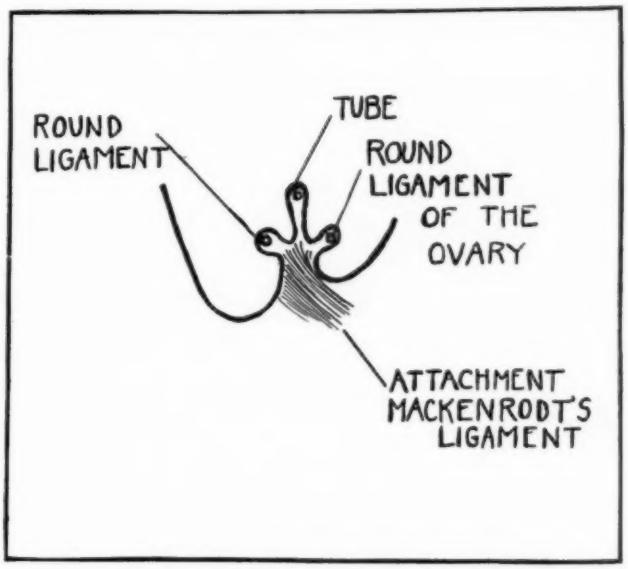
The main factors are tissue deficiency, childbearing, menopause and relaxation of the vaginal vault.

The cervix and uterus are held in position by the fibromuscular paravaginal and parametrial tissues sometimes called Mackenrodt's ligament. (Fig. 144A-B.) If this tissue is congenitally deficient or lacks resiliency it is readily stretched or torn during labor. As a result, the attachment of the upper third of the vagina and the lower third of the uterus becomes loosened, thus allowing the cervix and uterus to descend, producing a prolapse. In a patient whose parametrial tissue is potentially weak, the birth of one child may be sufficient to destroy what little resiliency was present and allow a prolapse to develop.

Childbearing *per se* is an etiological factor only in so far as it has to do with the stretching or loosening of the paravaginal and parametrial connective tissue. Prolapse is not dependent upon the number of children a woman has, but upon how these pregnancies affect her pelvic connective tissue. In Harlem Hospital where the incidence of colored patients is about 85 per cent, the number of prolapse cases seen is very small.



A



B

FIG. 144. A. Diagrammatic cross section at the level of the cervix. A-A shows fibromuscular tissue called Mackenrodt's ligament extending laterally towards pelvic wall. B. Longitudinal section (diagrammatic) through the broad ligament one inch lateral to the uterus showing how the fibromuscular tissue extends laterally at the base of the broad ligament.

They have frequent and difficult births but they have excellent fibromuscular connective tissue which does not become relaxed or loosened and this prevents the development of a prolapse.

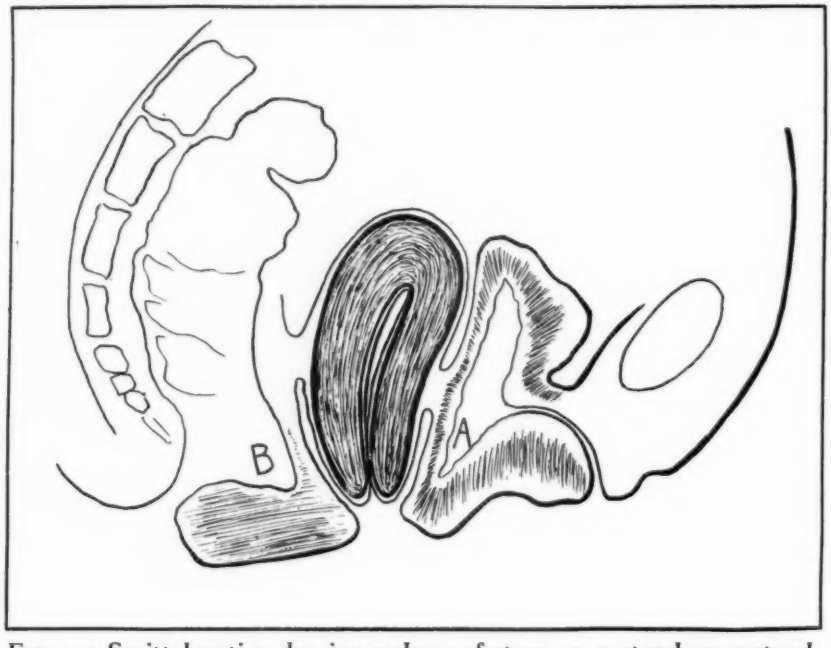


FIG. 145. Sagittal section showing prolapse of uterus. A, cystocele, B, rectocele.

Prolapse occurs most often in poor, overworked and frequently undernourished women who, having borne a child, cannot wait until proper involution has taken place before they must resume work. As a result the uterus remains retroverted, subinvoluted and heavy, drags on the parametrial ligaments which were none too strong at the start, and produces the first stages of prolapse.

The menopause may also have a definite bearing on the etiology of prolapse. A patient with weak fibromuscular parametrial tissue is a potential prolapse case. Although she may have been able to "carry on" and no prolapse develop because of the lack of an inciting cause, the onset of the menopause and its associated atrophy may be sufficient to allow a prolapse to occur.

When as a result of a stripping or tearing of the fascial attachments of the upper part of the vagina during labor, a relaxation of the vaginal vault occurs, the vault of the vagina

is allowed to roll out, producing a constant drag on the cervix and lower portion of the uterus with an ultimate stretching of the parametrial ligaments and prolapse.

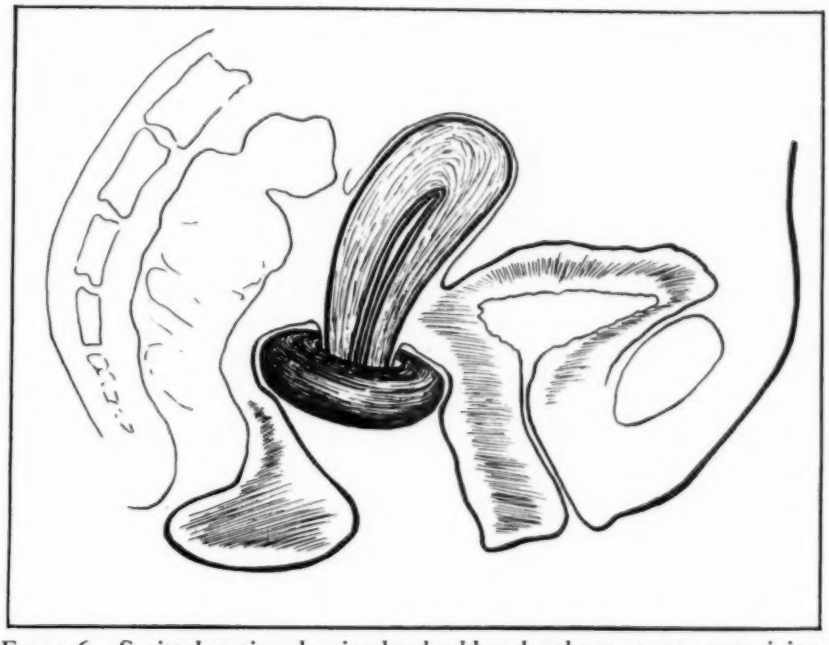


FIG. 146.—Sagittal section showing hard rubber doughnut pessary restraining the prolapse and supporting the cystocele and retocele.

SYMPTOMS

The prolapse itself causes few or no symptoms outside of a protruding mass, but some patients may complain of an aching or dragging pain in the lower back while standing, walking or working. Owing to the intimate relationship of the cervix to the bladder, a cystocele usually precedes and is nearly always found after the development of the prolapse. (Fig. 145.) The cystocele and the frequently associated relaxation of the urethral sphincter cause the patient definite discomfort. The cystocele may allow residual urine to remain in the bladder after voiding, thus causing bladder irritation, trigonitis and sometimes cystitis. The relaxation of the sphincter results in an inability to retain the urine, most evident on exertion, coughing, sneezing or straining.

Management of Prolapse. The cure of a prolapse, like the cure of any other condition in which fibromuscular tissue has been torn or stretched, is essentially surgical. wowever,

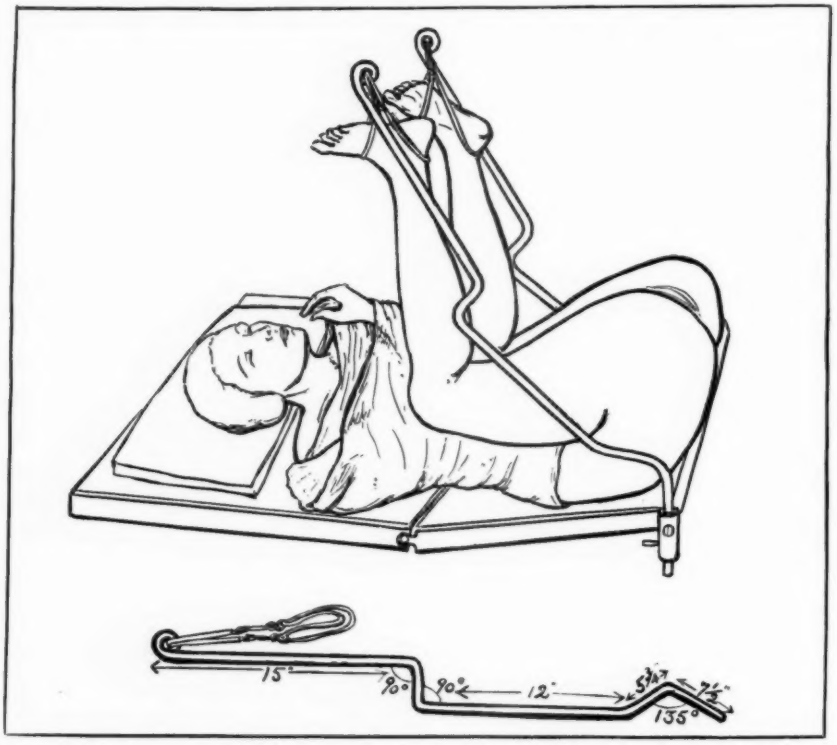


FIG. 147. Exaggerated lithotomy position with Jones' leg holders. This allows the assistants to stand in front of the legs so that they can be of more assistance and also allows them a better view of the operative field. Both legs must be elevated and lowered together, thus eliminating any back strain or backache. Insert shows angles and length of leg holders.

patients frequently refuse surgery, yet they require treatment. What can be done for them? The use of tampons gives temporary relief but they cannot be used over long periods of time. Hard rubber pessaries have a wide field of usefulness in prolapse cases. The pessary not only tends to hold the uterus forward, but also puts the vault of the vagina under tension and thus eliminates the constant downward drag on the cervix. It is invaluable in women during the childbearing period who desire more children before having a repair operation. (For the types of pessary, method of introduction, etc., see page 156.)

In women after the menopause the round hard rubber doughnut pessary is of considerable worth. The soft rubber pessary should never be used as it always becomes foul even

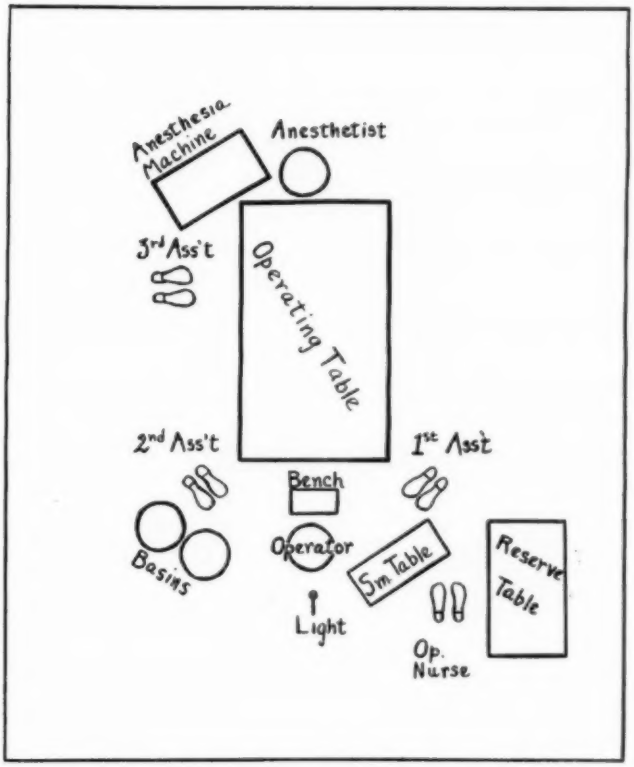


FIG. 148. Operating room set-up for all vaginal operations.

when given the best of care. Patients can be taught to insert the hard rubber pessary every morning and to remove it at night, thus preventing injury to the sensitive vaginal mucosa from the constant pressure of the pessary. It restrains the prolapse and may relieve the patient of some of her symptoms, particularly those referable to her bladder.

This pessary, if properly inserted, rests transversely in the vagina, supports the bladder anteriorly and aids in its complete emptying, thus preventing the collection of residual urine with its accompanying bladder irritation. (Fig. 146.) The cystitis and trigonitis when present should be treated (see Office Gynecology). Occasionally the prolapse cannot be retained by the use of the ordinary pessary due to complete lack of support

below, or to a rolling out of the entire vagina on the slightest strain. For these patients the Gellhorn pessary is very efficacious. Should this fail, some form of harness to hold the pessary in place may be required. This is mentioned only to be condemned, because it frequently produces ulcerations. When pessaries fail, surgery should be insisted upon.

DONT'S

1. Do not use a pessary unless the uterus has been replaced.

2. Do not use a pessary in the face of ulceration and edema of the projecting part.

3. Do not press the pessary anteriorly on inserting—exert all pressure on the perineal body.

Preparation of the Patient for Surgery. When the uterus has projected beyond the introitus for a long time it is usually markedly inflamed, congested, swollen and even ulcerated. In these cases, before operation can be considered, the uterus must be replaced and medical measures instituted to cure the ulceration and diminish the edema. Occasionally, owing to the marked inflammatory exudate and edema, replacement of the uterus may be difficult or even impossible. The patient must be put to bed and every effort made to replace the uterus. To reduce the inflammatory exudate and edema she should be given hot sitz-baths two or three times a day, absolute rest in bed with the foot of the bed elevated and wet dressings applied locally, between baths. If the uterus cannot be replaced, surgery is fraught with danger; the mortality is very high.

As soon as the inflammatory reaction has diminished sufficiently, the uterus is replaced and local treatments begun with the idea of further decreasing the edema of the tissues, healing the ulcerations, and restoring the parts to as nearly normal as possible before any operation is undertaken. For this purpose she is given hot douches twice daily followed by a boroglyceride of glycerine (25 per cent) tampon at night. Any ulcerated areas are treated with 3½ per cent tincture of iodine, or 5 per cent silver nitrate, to stimulate healing. During this period the gastrointestinal tract is regulated; the patient should

have a daily bowel movement, and for this purpose she should receive a tablespoonful of milk of magnesia every night, a tablespoonful of mineral oil every morning, and a soap suds enema every day if necessary.

If the prolapse is reducible and the patient cannot be hospitalized, a proper fitting pessary must be used which the patient should remove every night on retiring and reinsert every morning. The removal of the pessary is to be followed at night and its insertion preceded in the morning by a hot douche of one gallon of some mild antiseptic, such as iodine dram one to two quarts of water or 1–20,000 potassium permanganate. This is to be carried out until the patient is ready to enter the hospital.

Surgical Management. In the simple uncomplicated prolapse the preparation is the same as for total hysterectomy. All pubic hair is removed, a vaginal douche is given and the vagina, cervix and external genitalia are painted with Scott's solution or 4 per cent aqueous mercurochrome. This is repeated at least three times before the operation. The vaginal preparation in the operating room is the same as for any gynecological case. The vagina, perineum, pubis and thighs are thoroughly painted (Scott's solution) under vision and the patient is catheterized. She is then wheeled into the operating room and repainted before being draped.

OPERATIVE PROCEDURE

In young women who are desirous of retaining their procreative ability, the Manchester operation (amputation of the cervix, parametrial fixation, anterior and posterior wall repair) has given excellent results. In women beyond sex life the LeFort operation gives the best results with a minimal amount of surgery.

No attempt will be made to describe the various operative procedures one may use for the cure of prolapse. The interposition operation (Watkins-Wertheim operation) has given us our best results and will be described in detail. This operation is practically never performed alone. The associated pro-

cedures are (1) curettage, (2) amputation of the cervix, (3) resection of the tubes, (4) myomectomy or resection of the fundus, and (5) perineorrhaphy.

A curettage usually precedes the operation in order to eliminate the possibility of an early pregnancy or malignancy. The curettings must always be sent to the laboratory for examination. Associated with a prolapse of the uterus one frequently finds an elongated, hypertrophied, lacerated or diseased cervix which may have to be operated upon. If the interposition operation is performed, and the uterus with its cervix *is left more than three inches in length*, the cervix cannot be drawn far enough upward and backward, and as a result, the fundus will be pushed out of the vagina. If the operation is performed on patients who are still in the childbearing period they must be sterilized; the simplest procedure is resection of the internal end of the tubes. When the fundus of the uterus is delivered (Step 4) fibroids may be found and these may be removed by myomectomy. If the fundus contains many fibroids or if it is too large it may be resected, or a fundectomy performed. Following the interposition operation an existing rectocele or lacerated perineum should be repaired so as to restore the pelvic floor.

Set-up of the Operating Team. This is very essential for a smooth operation. This set-up can be used in all vaginal procedures. The patient is placed on the table in an exaggerated lithotomy position using the Jones' leg holders. (Fig. 147.) She is painted and catheterized in the anesthesia room, then repainted and draped in the operating room. A well illuminated vagina is essential; this can best be accomplished by the use of an overhead light and some form of floor flood light directed into the vagina above the surgeon's head. (Fig. 148.) The surgeon should be comfortably seated in front of the patient with a low flat bench for his feet which brings his knees level so that he can balance a small instrument tray on them. To the right of the operator should be a small table for sutures and reserve instruments, and to his left, a solution stand. The first assistant stands at the foot of the patient and

to her left, while the second assistant stands to her right, both facing the operator. The use of the Jones' leg holders allows the assistants to stand on the inside of the patient's legs and thereby be of more assistance to the operator. The sponge nurse or third assistant stands to the right of the patient behind the second assistant. This assistant usually has to stand on a low bench and lean over the patient, slightly, so that he is able to hold any instruments given him and pass such sponges or instruments as may be eecessary. The sponges and instruments used by the assistants are on a small tray or on the patient's abdomen. The instrument nurse stands to the right of the operator, behind the small table, and faces in the same direction as the operator. To her right is the large reserve instrument table. (Fig. 148.)

*Instrument Tray.** The instruments for use in the interposition operation are arranged on a sterile tray, a sterile towel is laid over these instruments and on this towel are placed the instruments for the curettage. A fairly small tray is used for this purpose so that it can be placed on the knees of the operator, making the instruments accessible to him and his assistants.

STEPS IN THE OPERATION

Step I. Exposure and Curettage. A weighted speculum is inserted into the vagina. The labia minora are stitched back to the thighs with a suture of No. 2 plain catgut cut in four, so as to keep them out of the field without using an assistant. (Fig. 149.) A small Deaver retractor is inserted anteriorly into the vagina exposing the cervix, which is grasped with a tenaculum forceps. The cervical canal is mechanically and chemically cleansed with a forceps holding a piece of one-half inch packing or the end of a gauze square dipped in full strength iodine solution. A uterine sound is introduced into the uterus and its length measured. *The uterus must never be left more than three inches long.*

*See FALK, H. C., Operating Room Procedure for Nurses and Internes. New York, 1934, G. P. Putnam's Sons.

The cervical canal is dilated with sound dilators dipped in iodine. An open sponge is placed flat on the weighted speculum in the vagina under the cervix so that all the material that

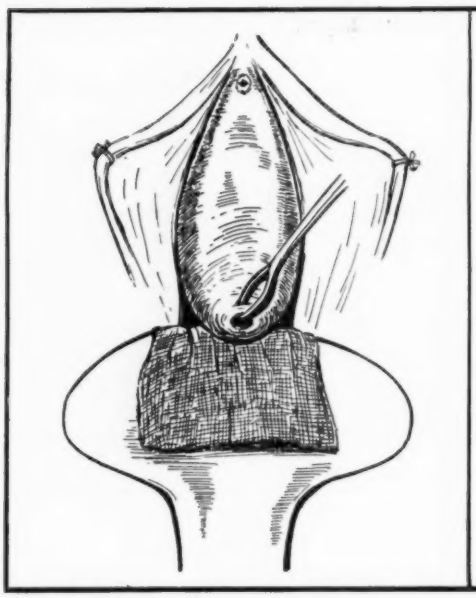


FIG. 149. Weighted speculum in the vagina, labia minora stitched back so as to be out of the field of operation, cervix grasped with tenaculum and an open gauze sponge placed under the cervix on the speculum to catch the curettings.

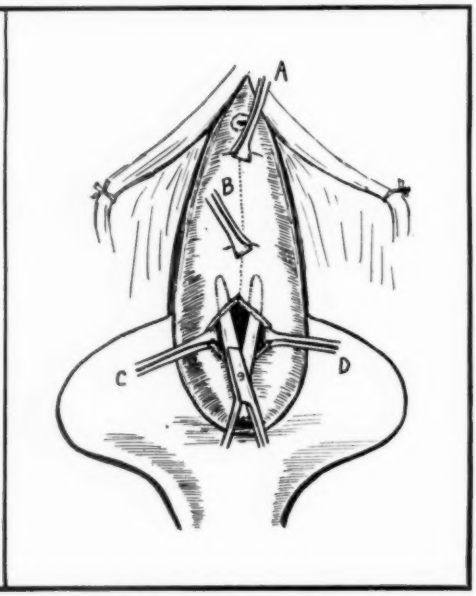


FIG. 150. Allis clamps A-B grasp vaginal mucosa above, clamps C-D hold cut edge of vaginal mucosa taut by downward traction. Scissors inserted into plane of cleavage before extending the incision upward to clamp B.

comes out of the uterus will be collected in the gauze. (Fig. 149.) The uterus is curetted and the curettings carefully saved and sent to the laboratory for examination.

The curettage instruments are lifted off the tray by the towel and given to the instrument nurse, thus uncovering the instruments for the interposition operation.

Step II. Separation of the Vaginal Mucosa. The cervix is held taut by the use of a bullet forceps or a Neuman tenaculum, and several Allis clamps are placed on the anterior vaginal wall, one above the other, about one inch apart. The last Allis clamp should be about one-half inch below the urethra so as to mark the upper limit of the incision in the anterior vaginal wall. With a knife a vertical incision is made in the midline

just through the mucous membrane, from the cervix to the first Allis clamp, which is removed. The vaginal mucosa on either side of the incision is grasped by an Allis clamp and the tenaculum on the cervix is removed. The vaginal mucosa is held taut by downward traction of these two Allis clamps. A curved Mayo scissors, closed, with its points directed away from the bladder, is introduced upward in the midline under the vaginal mucosa into a plane of cleavage, the blades are separated and withdrawn open. (Fig. 150.)

Surgeons frequently request information as to how to obtain the proper line of cleavage. It is very difficult to describe the exact approach for the proper line of cleavage. In separating, stick close to the mucous membrane and leave very little tissue on the flap. When the proper plane is reached the tissue is avascular and separated without much difficulty. There is such a thing as coming too close to the mucous membrane, but it is far better to come too close than to get too far away. Dr. Holden in describing his method of obtaining the proper line of cleavage with scissors states "snip, push and separate."

The incision through the mucous membrane is extended upward in the midline to the next Allis clamp which is removed, and the upper end of the cut vaginal mucosa on either side is grasped with an Allis clamp and the same procedure is repeated until the uppermost clamp is reached just under the urethra.

At this point the operator must decide whether or not an amputation of the cervix is to be performed or if the removal of the anterior lip of the cervix (Step VIII) will be sufficient. If an amputation of the cervix is to be done, a circular incision is made around the cervix through the vaginal mucosa as close to the external os as possible, yet far enough away so that all the diseased cervical tissue can be removed. (Fig. 152 insert.)

Separation of the Vaginal Mucosa from the Underlying Bladder. The cut edges of the vaginal mucosa on the patient's right side are grasped with four "T" clamps. The third assistant grasps the upper two clamps and the second assistant holds the lower two; these are held taut so as to create a flat surface. The first assistant presses a gauze square against the

bladder holding it taut, thus exposing the line of cleavage between the bladder and the vaginal mucosa which can be cut with a knife or scissors. (Fig. 151.) In dissecting the

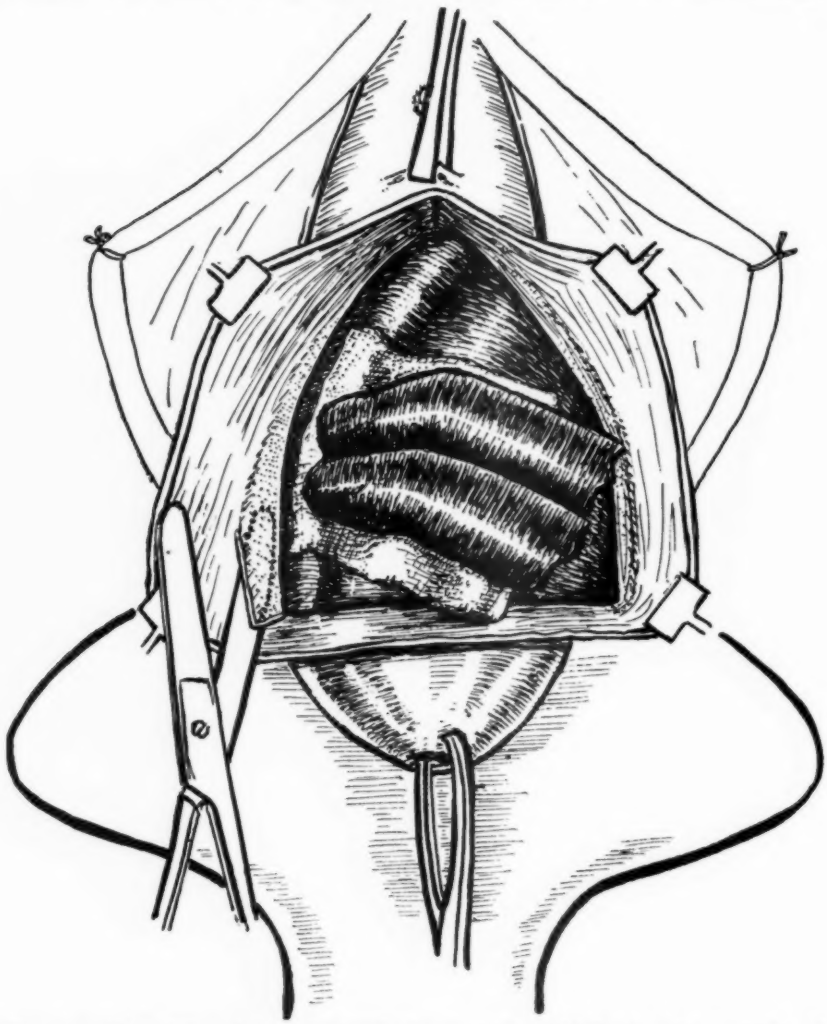


FIG. 151. The right edge of the vaginal mucosa is held flat by the second and third assistants—to simplify the drawing only two "T" clamps are shown. The first assistant presses on the bladder with a sponge as the operator finds the line of cleavage with the scissors.

vaginal mucous membrane away one should leave as much tissue on the bladder as possible. Many operators have discarded the interposition operation because of the bladder symptoms their patients have had following this procedure. This is due to the fact that they have stripped too much tissue

from the bladder and left the bladder wall too thin. It is essential in this operation *to leave as much tissue on the bladder as possible*, thereby eliminating post-operative bladder symp-

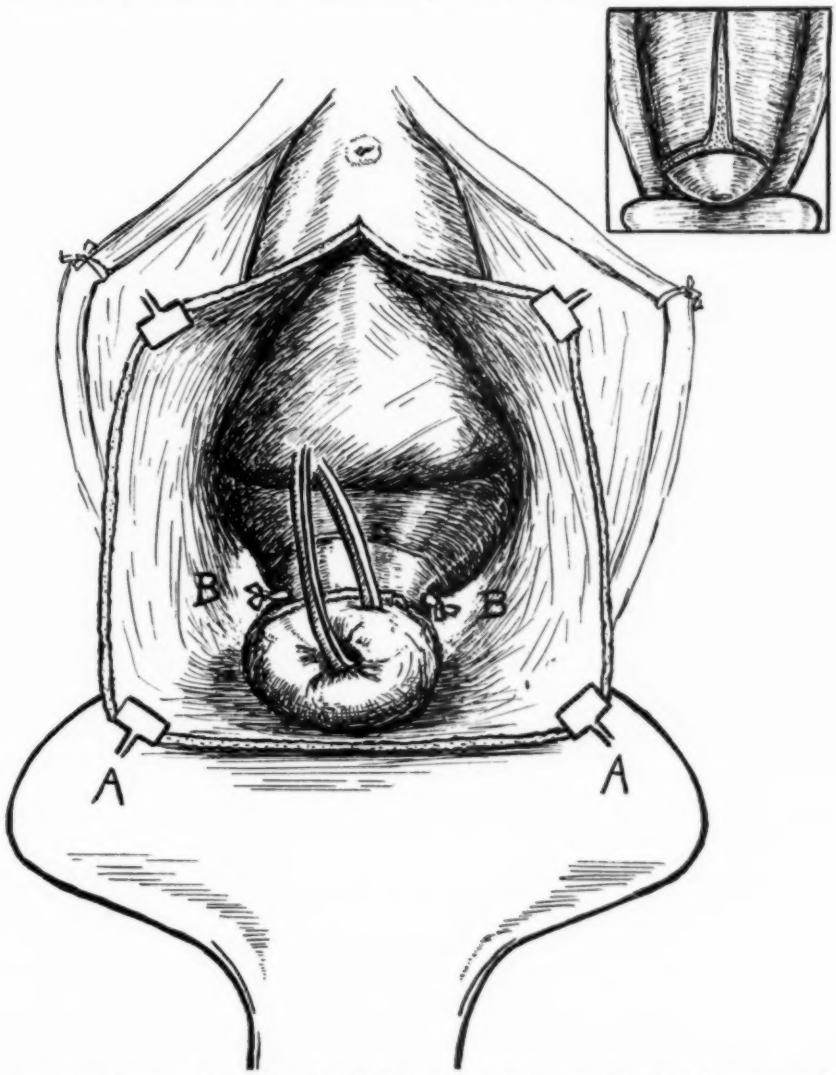


FIG. 152. Insert shows circular incision around the cervix. Cervix separated from vaginal mucosa preparatory to the amputation of the cervix. "T" clamps A-A hold edges of vaginal mucosa separated anteriorly, posteriorly and laterally. B-B heavy chromic catgut "crushing stitch" placed in either side of the cervix to control late hemorrhage.

toms. If the proper plane of cleavage is found, this separation is a fairly bloodless procedure. The separation should extend laterally far enough to expose the undersurface of the pubic

ramus. The same procedure is carried out on the left side. All bleeding points on the bladder should be clamped and ligated with fine catgut because the bladder will be placed

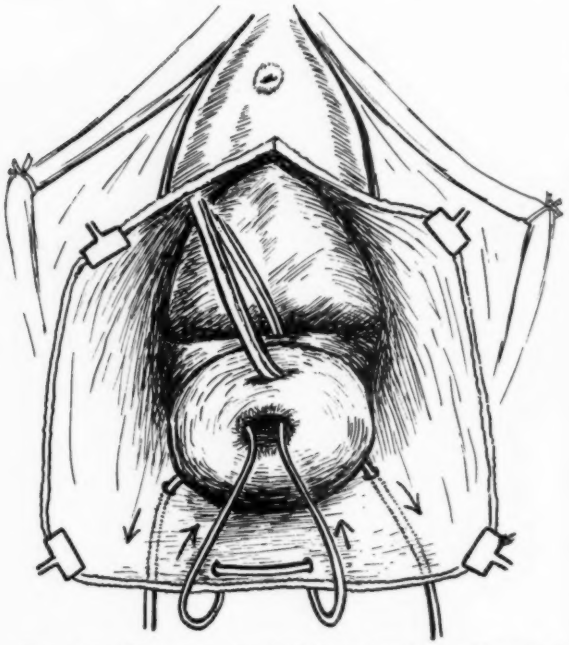


FIG. 153. Shows method of inserting inverting stitch after the cervix has been amputated.

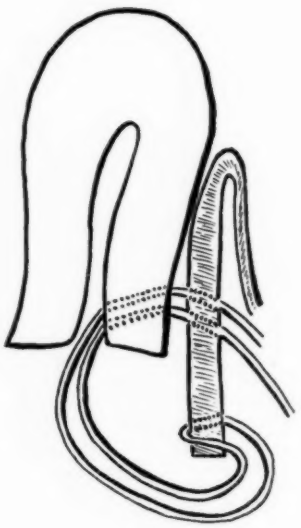


FIG. 154. Lateral view of inverting stitch.

in the peritoneal cavity upon completion of the operation. If these bleeding points are not carefully ligated, bleeding into the peritoneal cavity will produce post-operative morbidity.

If the cervix is to be amputated, the vaginal mucosa must be separated from the posterior and lateral walls of the cervix as far up as necessary so as to leave an ample flap for inversion. (Fig. 152.) If the cul-de-sac is opened in this procedure it should be closed with a plain catgut stitch. The mucous membrane having been freely separated anteriorly, posteriorly and laterally, one may now proceed with the amputation of the cervix. Before removing the cervix two procedures are carried out: first, 1 cc. of obstetrical pituitrin is injected directly into the cervix —this helps diminish the immediate hemorrhage; second, a suture of No. 3—40 day chromic catgut, cut in three on a cutting needle, is passed through the tissue on the lateral

aspect of the cervix just above the point where the amputation is to be made. (Fig. 152B.) This suture helps control the immediate and, more important, the late bleeding (ten day

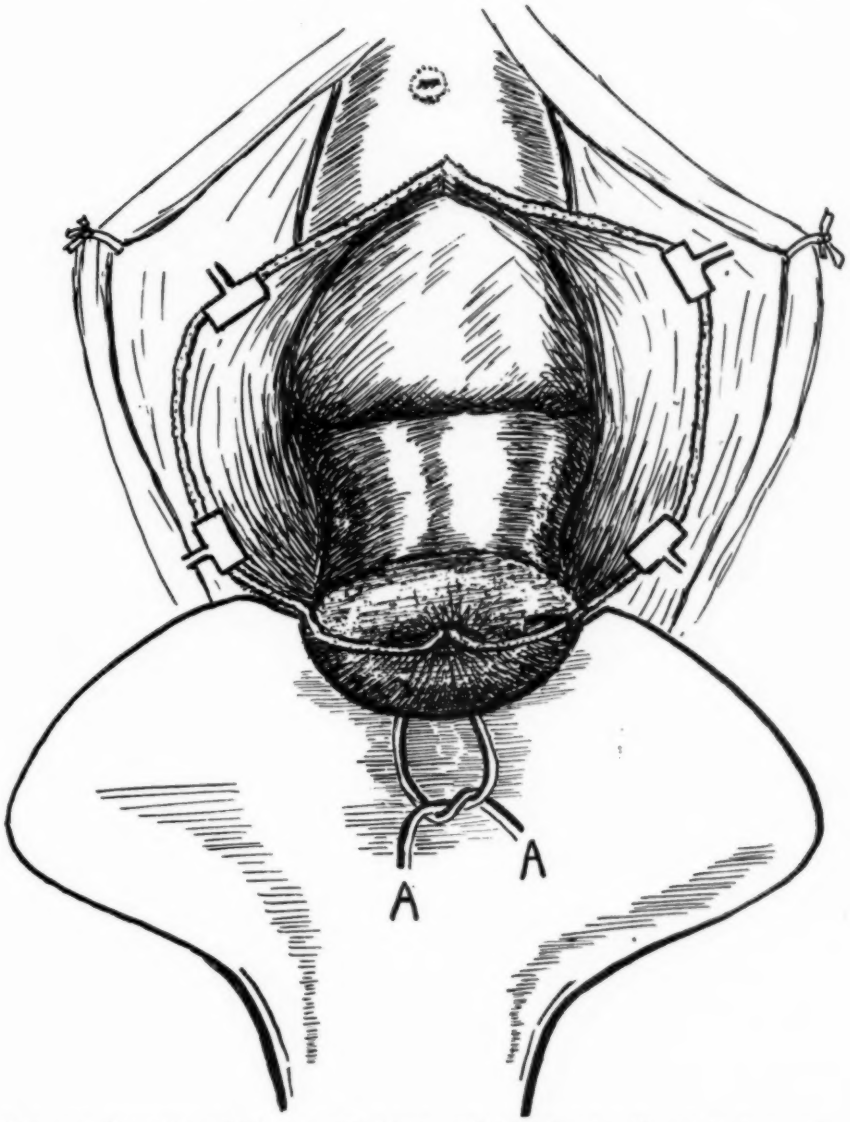


FIG. 155. Inversion stitch A-A pulled taut and tied, drawing the vaginal mucosa into the cervical canal and covering the posterior lip of the cervix.

hemorrhage). These sutures catch considerable tissue on the lateral aspect of the cervix and when tied should be pulled hard and long to act as a "crushing stitch." They should be

cut immediately because they control important vessels and should not be used for traction purposes. The cervix is cut across just below the two lateral chromic catgut sutures. If any

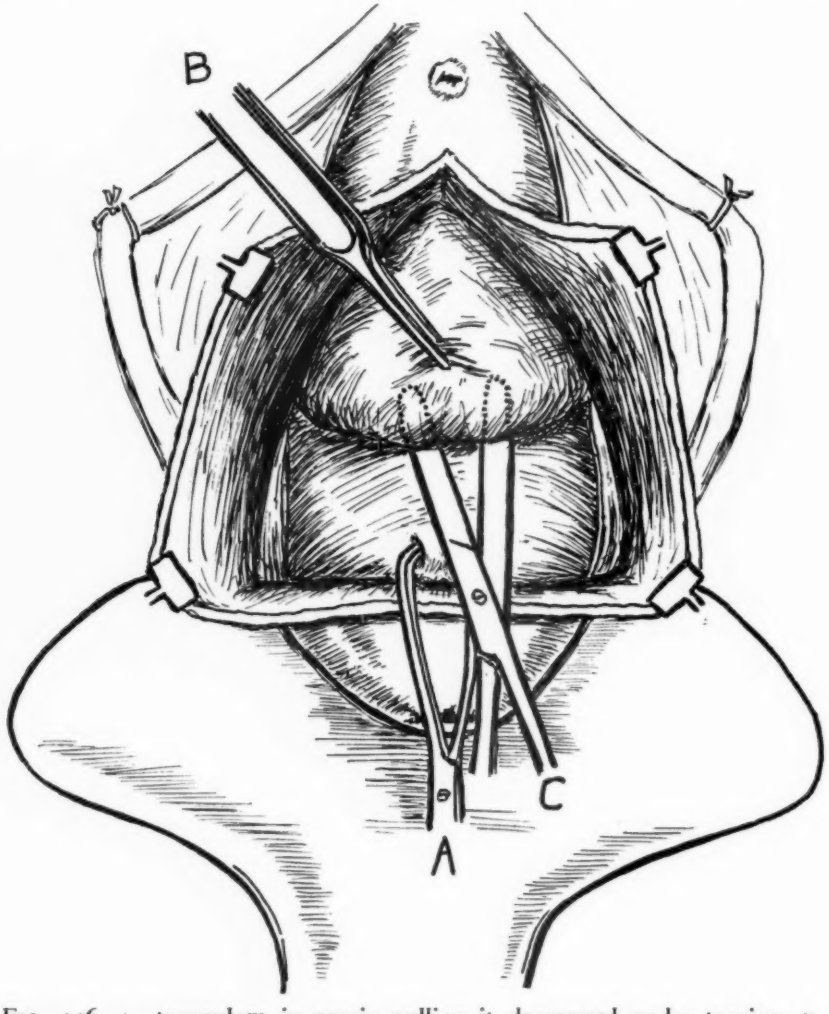


FIG. 156. A, tenaculum in cervix pulling it downward under tension; B, anatomical forceps lifting bladder; C, scissors bluntly introduced into the fold between the bladder and the anterior wall of the uterus, blades separated and withdrawn open.

active bleeding occurs in the cervix it must be controlled with fine chromic catgut sutures on a cutting needle. A hematoma should not be allowed to form under the mucous membrane flap, as it prevents healing and might cause late hemorrhage. The vaginal mucosa of the posterior lip is inverted into the

cervical canal with an inverting stitch (Figs. 153 and 154) which is left long and a Kut Klamp (p. 135) is applied for traction. (Fig. 155.)

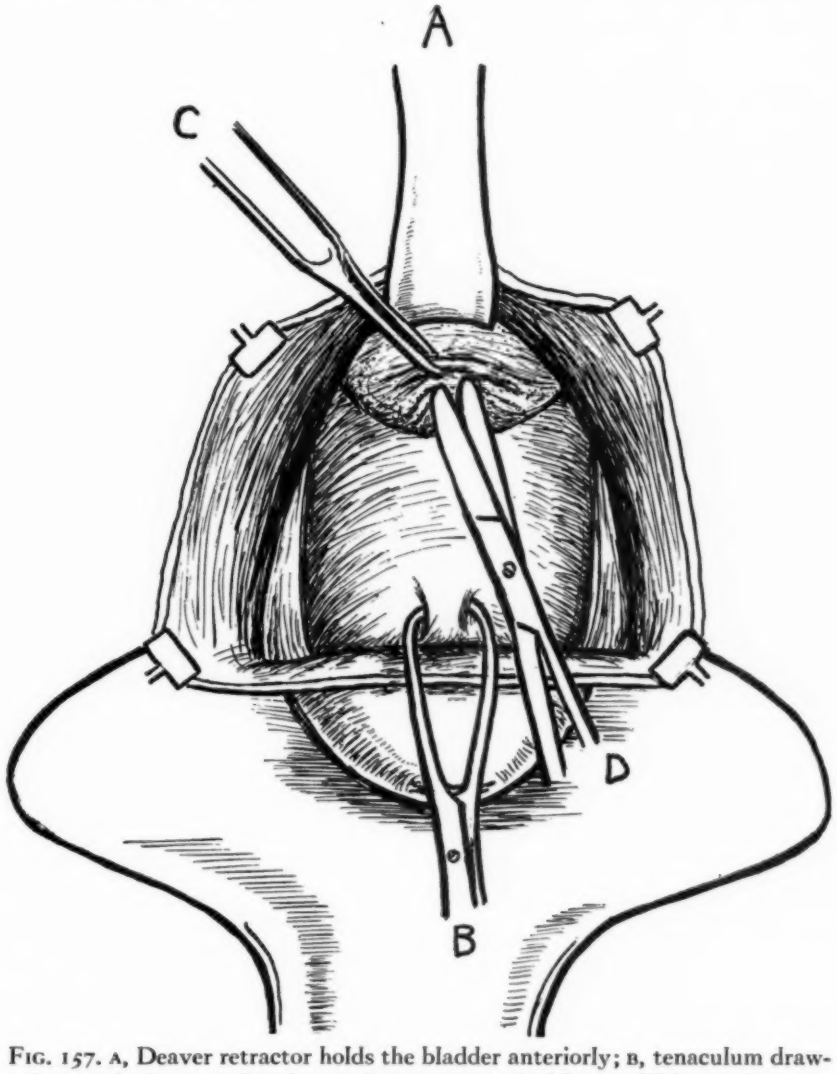


FIG. 157. A, Deaver retractor holds the bladder anteriorly; B, tenaculum drawing cervix downward under tension; C, anatomical forceps lifting peritoneum of vesicouterine fold; D, scissors opening vesicouterine fold.

Step III. Separation of the Bladder from Underlying Uterus and Opening of the Uterovesical Peritoneum. The cervix is grasped with a tenaculum and drawn downward under tension. This usually causes the fold between the bladder and the anterior wall of the uterus to become prominent. The bladder

is lifted with a smooth forceps and curved scissors with points upward, are bluntly introduced into this fold and the blades separated. The scissors are withdrawn open (Fig. 156), the

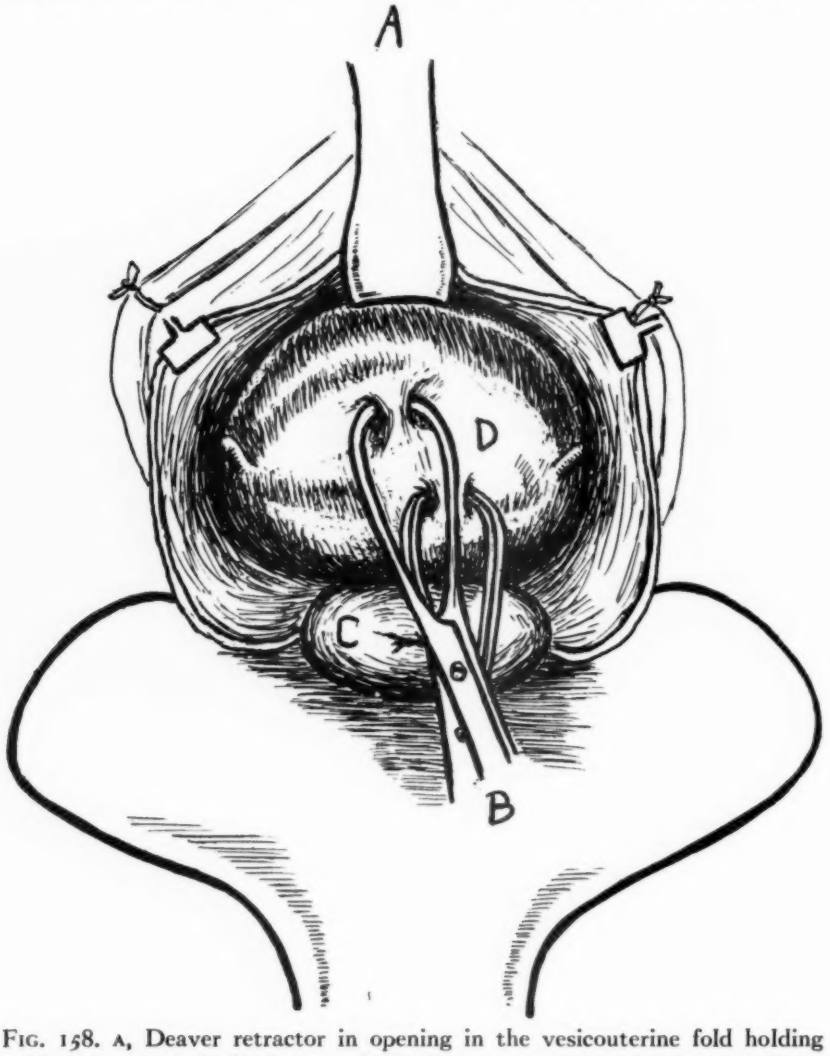


FIG. 158. A, Deaver retractor in opening in the vesicouterine fold holding bladder out of the field; B, two bullet forceps grasping D, fundus, and drawing through the opening in the peritoneum C, cervix.

finger is then introduced into this space and the bladder is bluntly pushed off the underlying uterus until the vesicouterine fold of peritoneum is palpated.

Caution. The most frequent error in technique, at this point, is to stay too close to the uterus for fear of entering the bladder. Bladder injury here, as in hysterectomy, is not

dangerous if recognized and closed in three layers. A retention catheter should be placed in the bladder to prevent any tension on the suture line.

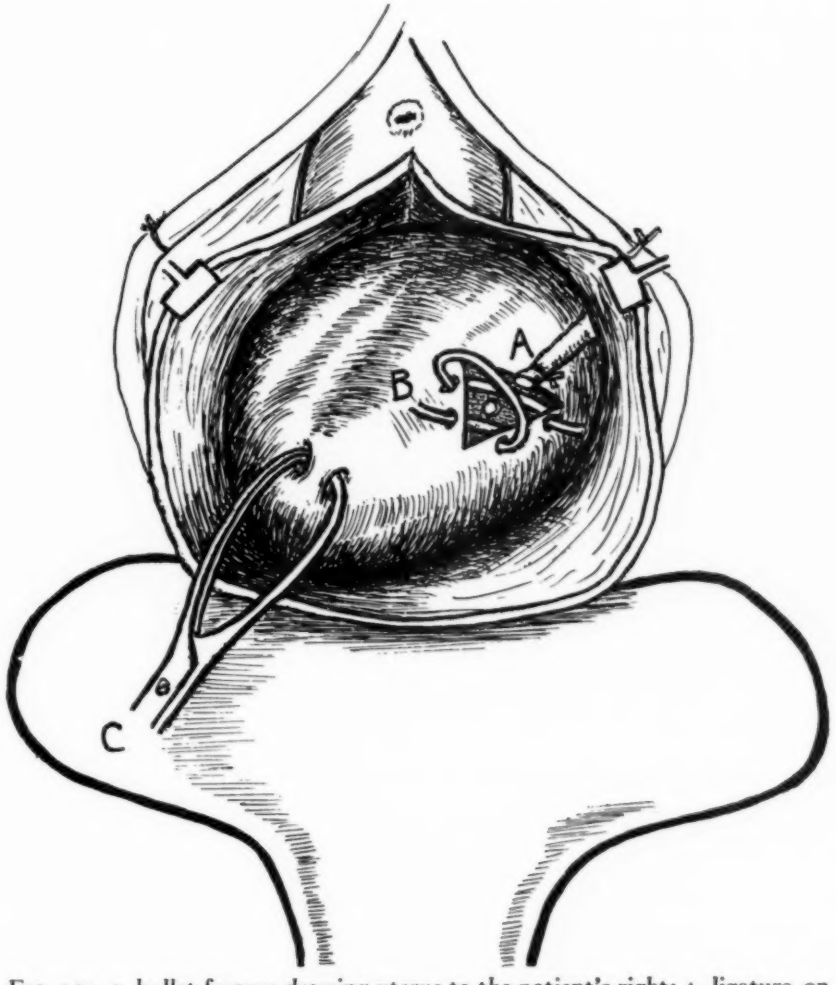


FIG. 159. C, bullet forceps drawing uterus to the patient's right; A, ligature on the tube and the tube cut across; B, figure of 8 stitch inserted into the cornu of the uterus after excising the uterine end of the tube.

When the proper line of cleavage is obtained, a small Deaver retractor is inserted anteriorly, lifting the bladder and exposing the vesicouterine peritoneum. (Fig. 157.) This peritoneum is grasped with a forceps and incised with a pair of curved scissors, the points of the scissors being towards the

uterus. A Deaver retractor is inserted into the peritoneum and the uterus is grasped with a small bullet forceps preparatory to delivery.

Step IV. Delivery of the Uterus. The anterior wall of the uterus is grasped by two bullet forceps (Fig. 158) one above the other, and the fundus is drawn through the opening. The tenaculum on the cervix is removed and all tension is removed from the cervix so as to allow the uterus to rotate on its transverse axis. As the cervix goes up the fundus comes down until it is delivered out of the peritoneal wound and into the vagina.

Caution. Do not make traction on the uterus with one bullet forceps—always use two, as one is apt to tear out. "Climb up" on the anterior wall of the uterus with several bullet forceps.

Step V. Tubal Resection—Fundectomy, etc. This depends upon several factors. If the patient is in the childbearing age it is necessary to sterilize her, as a patient with an interposition operation should not be allowed to become pregnant. To do this, the uterus is drawn to one side and a suture of No. 2 plain catgut cut in three on a Mayo needle, is passed under the tube and the tube ligated about three quarters of an inch from the cornu. The tube is cut across, leaving an ample amount beyond the ligature so as to prevent the tube slipping from the ligature. (Fig. 159.) The release of the tube itself is not so important as is the hemorrhage from the associated veins that would follow. The proximal end of the tube—that part inserted into the uterus—is excised with a wedge shaped incision. This opening is closed with a figure of 8 stitch of No. 2 plain catgut cut in three. The small piece of tube which has been excised is sent to the laboratory to be examined and the findings are recorded on the chart for purposes of record. The same procedure is carried out on the opposite tube.

Caution. Be sure to resect the tube and not the round ligament.

If, on delivery of the uterus, fibroids are found in the fundus they can be removed by myomectomy (p. 131) or by fundectomy (p. 100).

If the patient complains of bladder symptoms such as incontinence, bladder irritability, etc., the procedures to cure

these conditions are carried out at this time. The incontinence may be helped by Kelly sutures placed so as to narrow the urethra and tighten the sphincter of the bladder. (Fig. 160.)

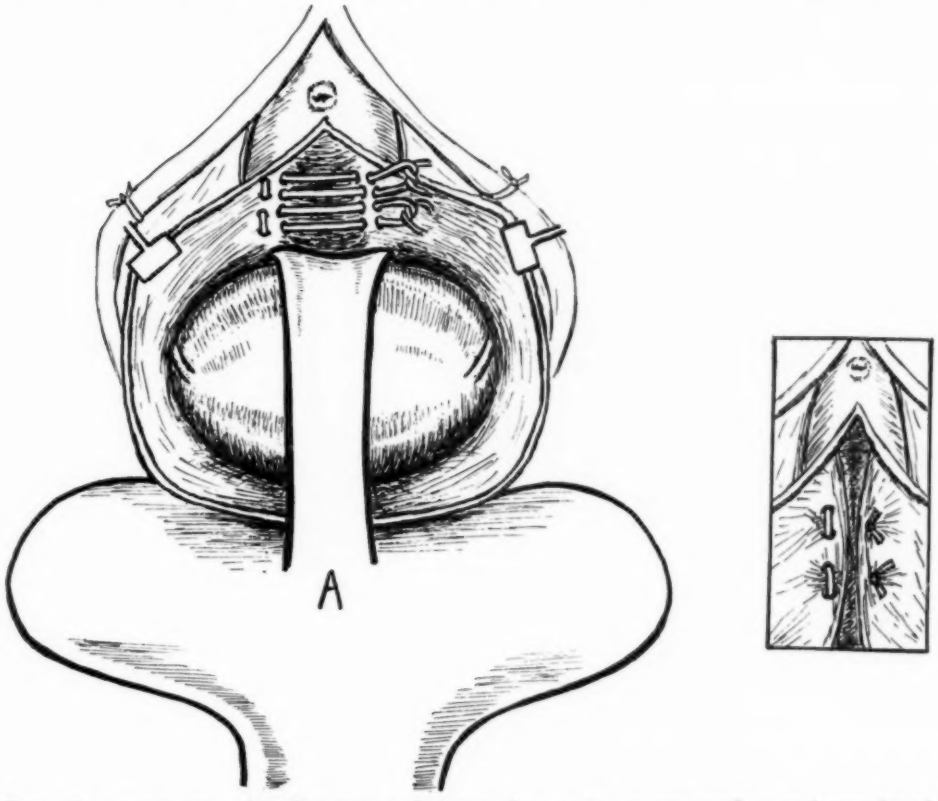


FIG. 160. A, retractor drawing uterus downward so as to expose urethra and associated portion of bladder. Mattress sutures inserted so as to narrow urethra and sphincter. Insert shows sutures tied.

Kelly stitches are mattress sutures inserted to either side of the urethra as far back on the bladder as one can go. As a rule, three such sutures can be inserted. The interposition operation, by placing the fundus of the uterus below the bladder, pushes the bladder upward, thus obliterating the pouch in which the retention has occurred. By curing the retention the associated irritability and cystitis are also cured.

Step VI. Insertion of Fixation Stitch. After all operative procedures on the fundus are completed, the fundus is fixed to the pubovesical fascia on the right and left sides by a double No. 3 chromic catgut suture, cut in two and tied double on a fairly small Mayo needle. These sutures are passed

from below upward through the fundus of the uterus, and from above downward through the pubovesical fascia, one on each side. To expose the pubovesical fascia a short Deaver

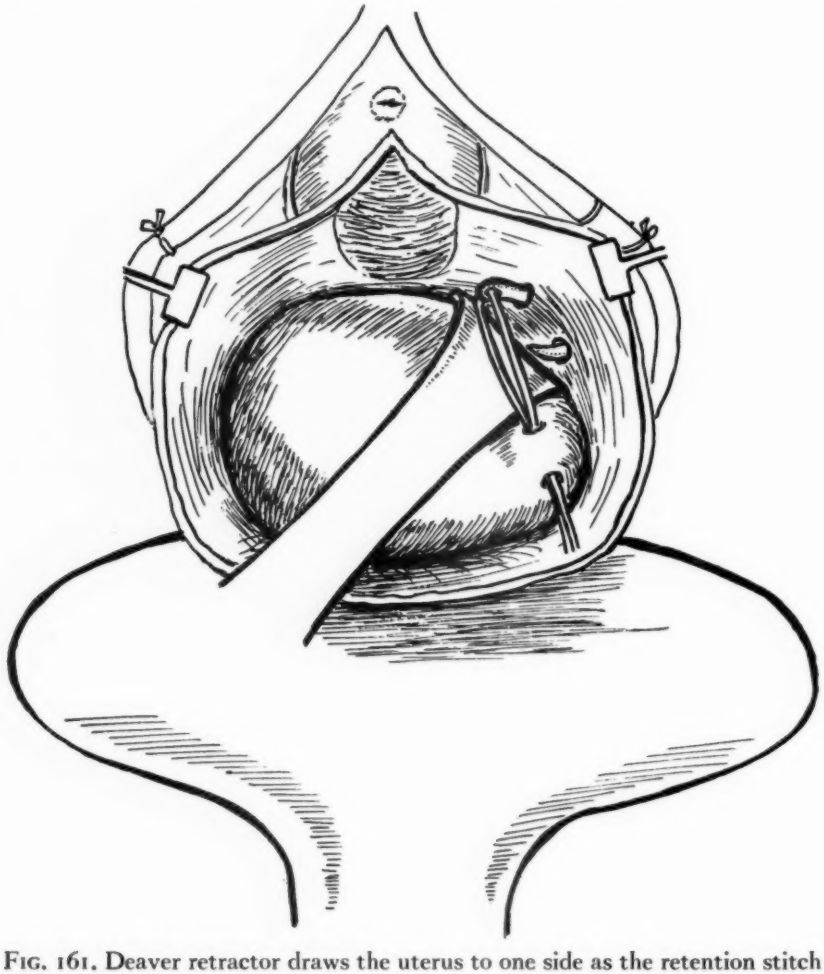


FIG. 161. Deaver retractor draws the uterus to one side as the retention stitch is inserted into the fundus from below upward and into the pubovesical fascia from above downward.

retractor is placed on the fundus of the uterus and it is retracted downward and medially. (Fig. 161.) These sutures are left long and a clamp is applied. They are not tied until Step VIII.

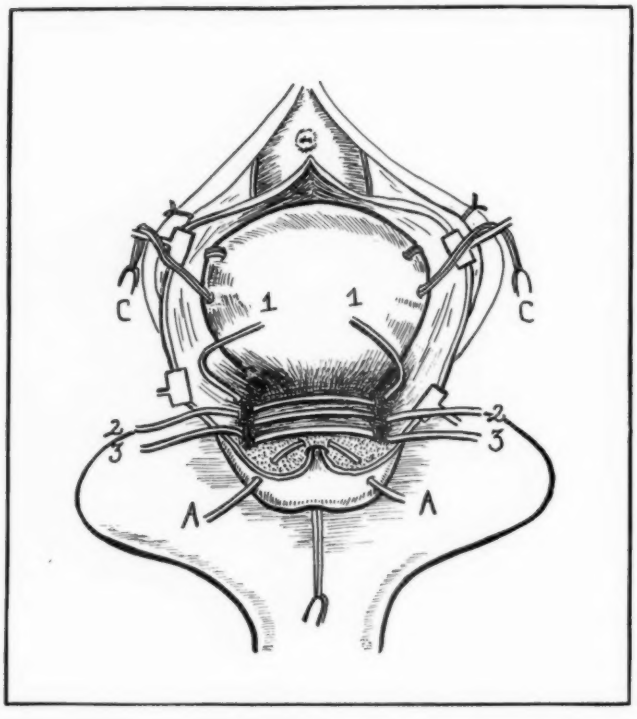
There are three points to be emphasized in the placing of these sutures: (1) they should be far enough laterally on the uterus so that when tied they do not make too much lateral

tension on the uterus and tear out; (2) they should be far enough upward on the anterior wall of the fundus so as not to make too great a bulge in the vagina; and (3) the sutures are introduced from below upward so that the knot when tied will lie on the outside of the uterus and not between the uterus and the pubovesical fascia.

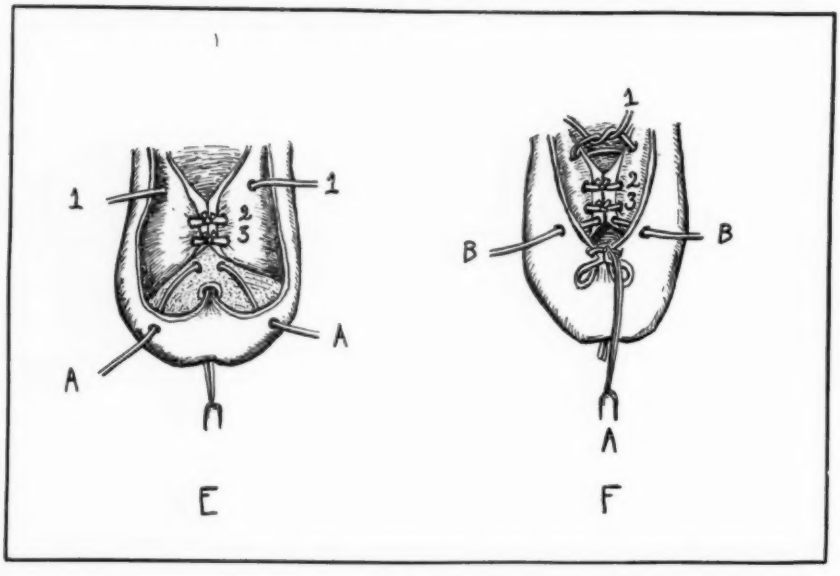
The patient is catheterized to rule out any bladder injury before the suturing of the vaginal mucosa is begun. If bloody urine is obtained the injury to the bladder is sought and when found is closed in three layers with fine chromic catgut and a retention catheter inserted.

Step VII. Cure of the Prolapse. The interposition operation *per se* does not cure the prolapse. To cure the prolapse it is necessary to bring the fibromuscular tissue on either side of the uterus in front of the cervix, thus shortening the stretched tissue and drawing the cervix upward and backward. To do this one of two procedures is carried out, depending upon whether or not an amputation of the cervix has been performed.

If the cervix has been amputated the fibromuscular tissue on either side of the uterus is brought in front of the cervix with a No. 2 chromic catgut stitch, cut in three on a Mayo needle. As a rule three sutures are used; these will be called *the deep sutures.* The first one is inserted at about the level of the internal os of the cervix, the second suture is just below this and the third is at the level of the external os. These sutures are clamped long and not tied until later. The redundant vaginal mucosa is cut "to suit," so as to bring it snugly in front of the cervix. No attempt is made to invert the anterior vaginal mucosa as was done with the posterior lip; the edges are simply brought together in front of the cervix and tied. In placing this suture a piece of the cervix is grasped so as to obliterate dead space. This suture is tied, cut and held long with a Kut Klamp. The third deep suture—the one at the level of the external os—is tied and cut short. The middle deep suture is also tied and cut short. (Fig. 162E.) The vaginal mucosa is closed in front of the cervix with interrupted stitches up to the level of the third



A



B

FIG. 162. The three deep sutures which will bring the lateral fibromuscular tissue (parametrial fixation) in front of the cervix are inserted No. 1 at the level of the internal os, No. 2 just below this and No. 3 almost at the level of the new external os. These sutures are clamped, left long and not tied until the first mucous membrane coaptation stitch A-A is inserted and tied. C-C retention stitch to pubovesical fascia. Insert E parametrial fixation sutures No. 3 and No. 2 are tied, mucous membrane stitch A-A is about to be tied. There is no attempt made to invert the mucous membrane on the anterior lip as was done on the posterior lip Fig. 155. Insert F, parametrial fixation stitch No. 1 is tied. Suture B-B closing the mucous membrane inserted and tied.

deep stitch which is tied and cut short. As each of the deep sutures is tied, it draws the cervix further upward and backward. As each vaginal stitch is inserted and is about to be

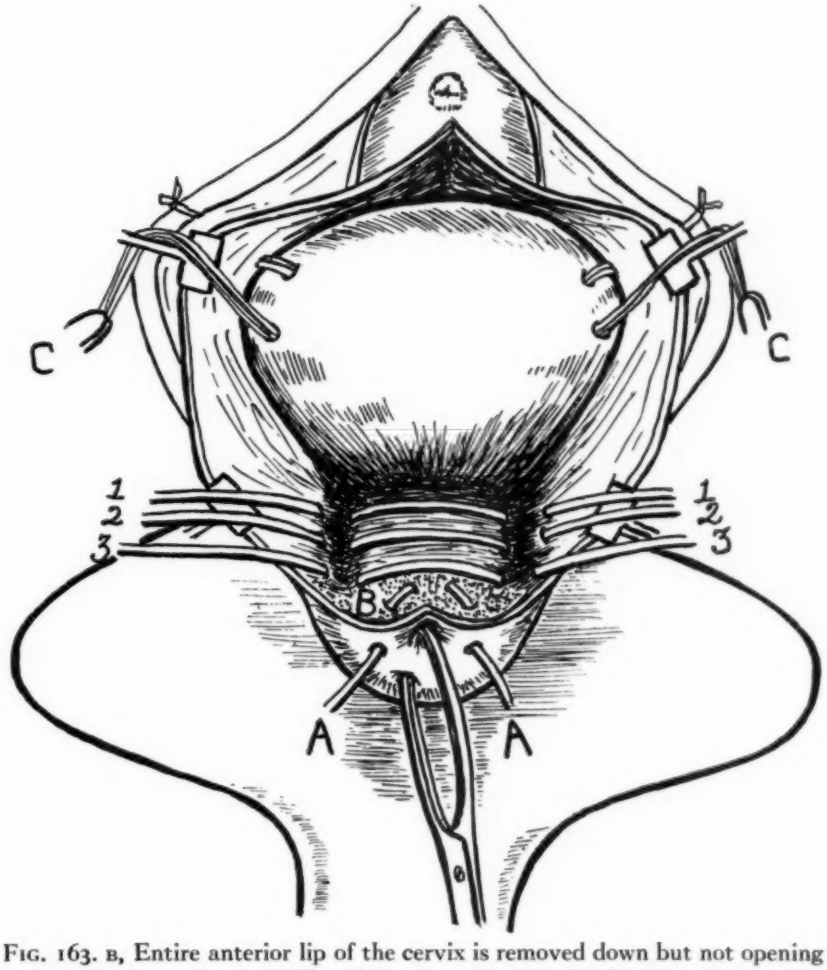


FIG. 163. B, Entire anterior lip of the cervix is removed down but not opening into the cervical canal. The three deep parametrial fixation sutures No. 1-2-3 are inserted, clamped long and not tied. A-A stitch to close mucous membrane and bring cut edges of anterior lip of the cervix in coaptation. C-C retention stitch to pubovesical fascia.

tied, the preceding one which has been used as a traction stitch is cut. The cut edges of the vaginal mucosa are united with interrupted sutures of No. 2 chromic catgut, cut in four on a Mayo needle, up to about the middle of the anterior wall of the uterus; each suture catches a small piece of the anterior

wall for fixation as well as obliteration of dead space. *The deep and superficial sutures over the cervix force it upwards and backwards and hold it there, thus curing the prolapse.*

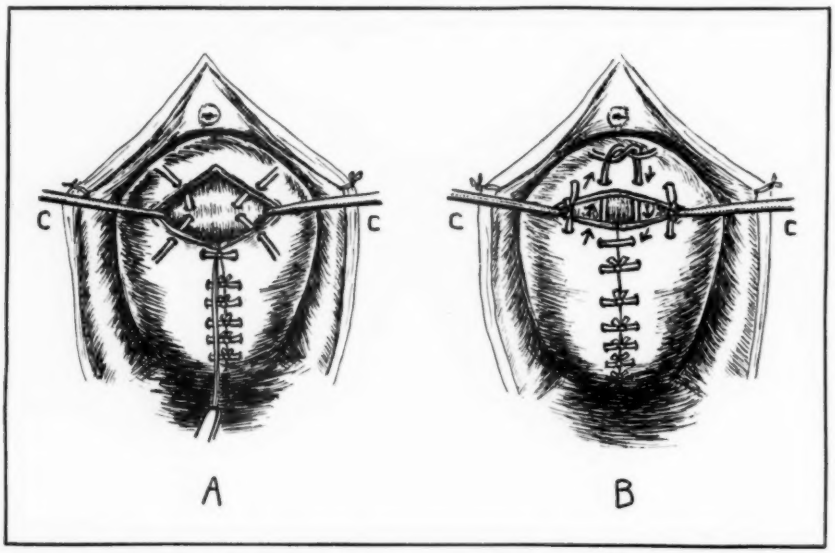


FIG. 164. A, redundant vaginal mucosa excised; c-c skin hooks inserted to draw opening laterally; sutures inserted from above downward grasping a piece of the anterior wall of the uterus to obliterate dead space. B, insertion of "U" stitch to close opening in vaginal mucosa. c-c skin hooks holding wound taut.

If no amputation of the cervix was performed the fibromuscular tissue lateral to the cervix is brought together in the following manner. The cervix is grasped on either side with a bullet forceps and the anterior lip of the cervix is removed down to the mucosa of the cervical canal which is not opened. (Fig. 163.) This removes part of the anterior vaginal wall and the anterior lip of the cervix, thus exposing the fibromuscular tissue lateral to the uterus.

The same three deep sutures are inserted into the lateral fibromuscular tissue beginning at the level of the internal os and extending downward to the external os. These sutures grasp the fibromuscular tissue and a piece of the cervix on each side so as to diminish the size of the cervix as well as to shorten the stretched fibromuscular tissue. The redundant vaginal mucosa is cut "to suit" and it is closed with interrupted catgut sutures up to the middle of the anterior uterine wall.

Step VIII. Tying of the Fixation Sutures. Before closing the rest of the anterior vaginal wall, the two fixation stitches inserted in Step VI are tied and cut short.

Step IX. Elimination of Urethrocele. Very frequently the patient has a redundant urethrocele. To eliminate this pouching, two skin hooks are placed, one on each side of the redundant mucous membrane (Fig. 164), the excess mucous membrane is removed and the cut edges sutured from above downward. The last stitch is inserted as a "U" stitch and tied, closing the entire area.

Step X. One half ounce of 4 per cent aqueous mercurochrome is introduced into the bladder. This causes an aseptic cystitis with urinary frequency and diminishes the necessity of post-operative catheterization.

CYSTOCELE

Cystocele is a prolapse of the bladder towards the vaginal outlet. It rarely occurs alone; it is usually associated with a lacerated perineum and a beginning descent of the uterus. Cystocele may result from either a childbirth injury, or a congenital weakness of the tissues in a nulliparous patient. A considerable amount of literature has accumulated with reference to the presence or absence of a fascia between the vagina and bladder, and as to whether or not a rupture of this fascia with a herniation of the bladder through the opening is the basic etiological factor in a cystocele. No attempt will be made in this discussion to prove or disprove any of these contentions. This discussion will be limited entirely to the practical methods of handling the condition.

The symptomatology of this condition is very similar to that of prolapse of the uterus because it is the descent of the bladder that is responsible for most of the symptoms of uterine prolapse. A bearing-down pain, feeling of fatigue and pressure may be present. However, the bladder symptoms, frequency, burning, etc., due to incomplete emptying of the bladder usually bring the patient for treatment. In patients at or near the menopause, owing to a relaxation of the bladder sphincter,

there may be a partial incontinence and loss of urine on coughing, sneezing or laughing.

Treatment. Medical therapy consists of treating the associated trigonitis or cystitis, and the use of a proper fitting pessary, either of the hard rubber doughnut variety, which the patient introduces every morning and removes every night, or of the Smith or Gellhorn type of pessary. Pessaries frequently fail due to the associated lesions, particularly a lacerated perineum. This produces a lack of support on the part of the pelvic floor, making it impossible for the pessary to be held in its proper place.

Surgical. The principles laid down by Hadra of Texas in 1889 for the cure of cystocele still apply.

1. Detachment of the bladder from the anterior vaginal wall medially and laterally and from the anterior wall of the uterus sufficient to allow the bladder to be displaced upward into the pelvis.

2. Resection of the vagina to remove excess tissue.

3. Suture of the anterior vaginal wall high enough on the anterior wall of the uterus to prevent the descent of the bladder.

The first step in the procedure allows the bladder to be displaced upward, whereas the second and third steps, by removing excess vaginal tissue and shortening the lateral ligaments in front of the cervix, draw the cervix upwards and backwards and correct the tendency of the uterus to prolapse, and thus prevent a recurrence of the cystocele.

A disregard of the close relationship between cystocele and prolapse and their frequent occurrence simultaneously has been the cause of the many recurrences following cystocele operation.

The interposition operation (Watkins-Wertheim) is also used for the cure of a cystocele, particularly in patients late in sex life or those in whom the prolapse is the more prominent symptom. The interposition operation should not be used in young women without a thorough understanding on the part of both husband and wife that further pregnancies cannot occur, as the tubes must be resected in all patients with an

interposition in the childbearing age. In the interposition operation the uterus is placed below the bladder and supports it.

The operating room set-up, instruments, steps in the operation, etc., for cystocele are practically the same as those in interposition except that the vesicouterine pouch is not opened and the uterus is not brought down in front of the bladder.

Step I. Dilatation and Curettage.

Step II. Separation of the Anterior Vaginal Mucous Membrane. This is the same as in interposition except that it does not extend so far laterally.

Step III. The bladder is separated from the underlying uterus in the same way as in the interposition operation (Fig. 156), but the vesicouterine fold of peritoneum is not opened.

Step IV. This is practically the same as Step VII in the interposition, where the cervix has not been amputated. Three deep sutures are inserted and the lateral fibromuscular tissue is brought together in front of the cervix. The first of these deep sutures may go through the muscular wall of the bladder so as to displace and fix it upward on the anterior uterine wall.

Step V. Excision of Redundant Vaginal Mucosa and Closure of the Anterior Vaginal Wall. The excess of vaginal mucosa is removed "to suit" and the cut edges are approximated by interrupted chromic catgut sutures beginning at the cervix and extending upward to the entire extent of the wound. As these sutures are inserted, the lowermost deep sutures of Step IV are tied and cut short in the same manner as in the interposition operation.

Caution. Be careful in the cystocele operation not to shorten the anterior vaginal wall. If this is done the cervix is drawn forward under the symphysis, and a retrodisplacement of the uterus is produced which may produce more distressing symptoms than the original cystocele. Following the operation, one half ounce of 4 per cent aqueous mercurochrome is introduced into the bladder. This causes an aseptic cystitis with urinary frequency and diminishes the necessity of post-operative catheterization.

PERINEORRHAPHY

Perineal lacerations as a result of non-obstetrical trauma do occur but are relatively rare. In any labor, whether primiparous or multiparous, there is a marked stretching of the perineum, producing lacerations either subcutaneously or through the mucous membrane. More and more, obstetricians are using either a median or lateral episiotomy to prevent the over-stretching of the tissues; this also permits a better primary perineal repair. However, in spite of all precautions and with the best obstetrics, irregular lacerations of the perineum do occur. These should, whenever possible, be repaired immediately at delivery or within twenty-four hours.

A good primary repair requires that the approximation of the tissues be as near their original relationship as possible. To do this requires good light and adequate assistance. A proper approximation of the deep fascial and muscular structures is far more important than a correct closure of the rent in the vaginal mucosa. To prevent infection, particularly in postpartum surgery, one must augment the usual aseptic technique with an antiseptic technique. This is done by flushing the wound two or three times during the operation with either 3½ per cent iodine solution or 2 per cent mercurochrome-acetone-alcohol solution (Scott's), or whatever antiseptic is being used. Should a primary repair or a repair within twenty-four hours not be performed, the operation is best delayed until complete involution of the tissues has taken place; this requires approximately nine months.

There are four types of laceration and relaxation of the posterior vaginal wall: (1) enterocele, (2) rectocele, (3) incomplete perineal laceration, (4) complete perineal laceration.

Enterocele is a true hernia through the cul-de-sac and through the upper part of the vagina.

Rectocele usually results from a subcutaneous stretching or tearing of the supporting structures in front of the rectum. It is a herniation of the rectal wall through this break in the fibromuscular structure of the rectovaginal wall.

Incomplete perineal laceration is a laceration of the fibromuscular tissue of the pelvic floor—levator ani muscle.

Complete perineal laceration is a laceration through the entire pelvic floor, involving the sphincter ani muscle with or without laceration of the anterior rectal wall.

A rectocele or enterocele without a lacerated perineum is infrequent; the opposite, however, is not always true. A complete laceration of the perineum can and does occur without a rectocele.

SYMPTOMATOLOGY

Simple laceration of the perineum rarely gives symptoms. When a rectocele is present there is a protrusion of the posterior vaginal wall (anterior rectal wall) on straining at stool. Occasionally a sacculation of feces in the rectocele causes symptoms and movement of the bowels may require pressure from within the vagina. Rectocele in its pathology and sacculation, simulates cystocele (herniation of the bladder through the anterior vaginal wall).

In complete laceration of the perineum the sphincter ani muscles, internal and external, are torn. They are the primary means of controlling and retaining fecal contents. In the absence of function of these muscles it is remarkable what a properly conditioned levator ani muscle can accomplish. Many of these patients with complete lacerations are able to control constipated movements, but most of them are unable to control diarrheal movements. Most of them cannot control the passage of gas, and these gases frequently carry away fecal contents and moisture, so that they are required to wear a pad constantly.

Primary Repair of Perineal Lacerations. Incomplete lacerations can and should be repaired immediately, either by through-and-through sutures, using a nonabsorbable material, or layer by layer approximation, using chromic catgut.

If a complete laceration occurs in the home it is far better to take the patient to the hospital for the repair than to do it in the home with poor assistance and usually very poor light.

The recognition of swollen, torn tissue immediately after delivery is extremely difficult. Any attempt to approximate these tissues is not easy even under the best conditions. It is usually better to wait until the next morning, after the patient has had a rest and the obstetrician has had some sleep, before attempting the primary repair of a complete tear.

After delivery of the placenta, one large piece of gauze should be inserted in the vagina to prevent the uterine bleeding from clouding the operative field. This gauze should be soaked in some antiseptic. The wound edges should be well exposed (Gilpi retractor—small Deavers). The rectal mucous membrane is exposed and sutured with silk, the knots being tied toward the rectum. After closure of the rectum, the wound is washed with an antiseptic solution and the operator's gloves are changed. The tissue between the vagina and the rectum is approximated with fine interrupted chromic sutures. The vaginal mucosa is closed with interrupted chromic catgut sutures, the knots being towards the vagina. Whenever possible the suture layers, vaginal and rectal, should not be superimposed. The wound is washed with an antiseptic. The rectal sphincter is then sutured to its fellow on the opposite side with two or three interrupted chromic sutures. The perineal muscular tissue, levator ani, etc., are closed in layers. If this cannot be done, the perineal wound may be closed with through-and-through sutures, care being taken to bring the anal edges in coaptation, using a nonabsorbable suture material.

Post-operative Care. Some men state that no special care need be taken with regard to the regulation of the bowels after a primary repair of a complete laceration. This cannot be accepted. These patients should be constipated for a period of several (seven to eight) days and then their bowels should be made to move very gently. *There must be no straining at stool.* The use of licorice powder or mineral oil by mouth and several ounces of olive oil inserted into the rectum through a small tube daily after the seventh day, in order to keep the bowels soft and prevent the development of constipated stool masses, is advisable.

If a careful coaptation of the torn tissues under aseptic and antiseptic precautions was performed, the probability of obtaining a cure is excellent. The percentage of primary cures where the laceration is recognized and properly treated is very high.

If the primary repair has been followed by failure, what is the next procedure? Under no circumstances should further surgery be attempted on the patient for at least nine months.

Whenever secondary plastic work on the perineum has been carried out in anything under nine months, the results have been uniformly bad. The patient's tissues tear readily, there is an excessive amount of hemorrhage and the tissues do not heal well. If possible, one should wait one year, as it takes the tissues a long time to involute properly and return to a normal state.

What can be done for these patients during the period of waiting? In the incomplete lacerations nothing need be done. Patients with complete lacerations should be taught to keep their bowels thoroughly clean, using an enema very frequently. As far as possible, they should stay on a constipating, low residue diet. Hot vaginal douches and sitz-baths should be taken frequently to reduce the congestion and edema of the tissues. They should return for re-examination at fairly regular intervals; this is done mainly for the purpose of keeping contact with the patient and helping her maintain her morale during this trying period.

Late Repair of Lacerations of the Perineum. In all perineal plastic surgery certain points in technique are of paramount importance.

1. The reconstruction of the fascial-muscular tissue to as nearly a normal position as possible—overcorrection with subsequent dyspareunia is worse than undercorrection.

2. Tissues must be brought together without tension. Sutures should not be tied too tightly, otherwise included tissue will die and become hard dense scar tissue. *Tissue sutures are placed for coaptation only.*

3. All scar tissue should be excised so as to restore a pliable though firm perineum. If thick inelastic bands of scar tissue are allowed to remain, dyspareunia may result.

Simple Lacerations of the Perineum—Perineorrhaphy. This procedure is usually performed as the last step in a series of procedures. If it is the only vaginal plastic operation that is to

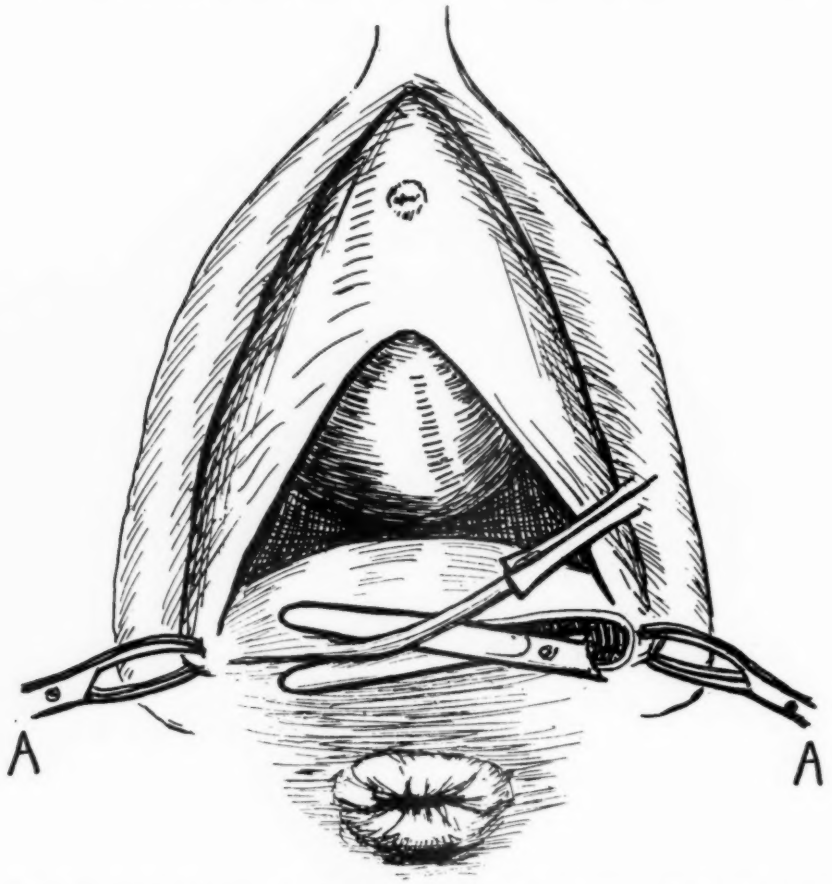


FIG. 165. Bullet forceps A-A placed at the edge of the carunculae myrtiformes and the mucocutaneous junction is held taut and excised with a scissors.

be performed, it has been found of great help to do it after the laparotomy. In this way the operator and his staff, may stay clean and use the same instrument set-up without any loss of time or re-scrubbing.

Operating Room Set-up. As for any vaginal operation. (Fig. 148.)

Step I. Excision of Mucocutaneous Junction. With two small bullet forceps (small tenaculum) the two lateral edges of the caruncula myrtiformes are grasped and held taut. The

superficial tissue, skin and mucous membrane at the mucocutaneous junction is excised from one tenaculum to the other. (Fig. 165.)

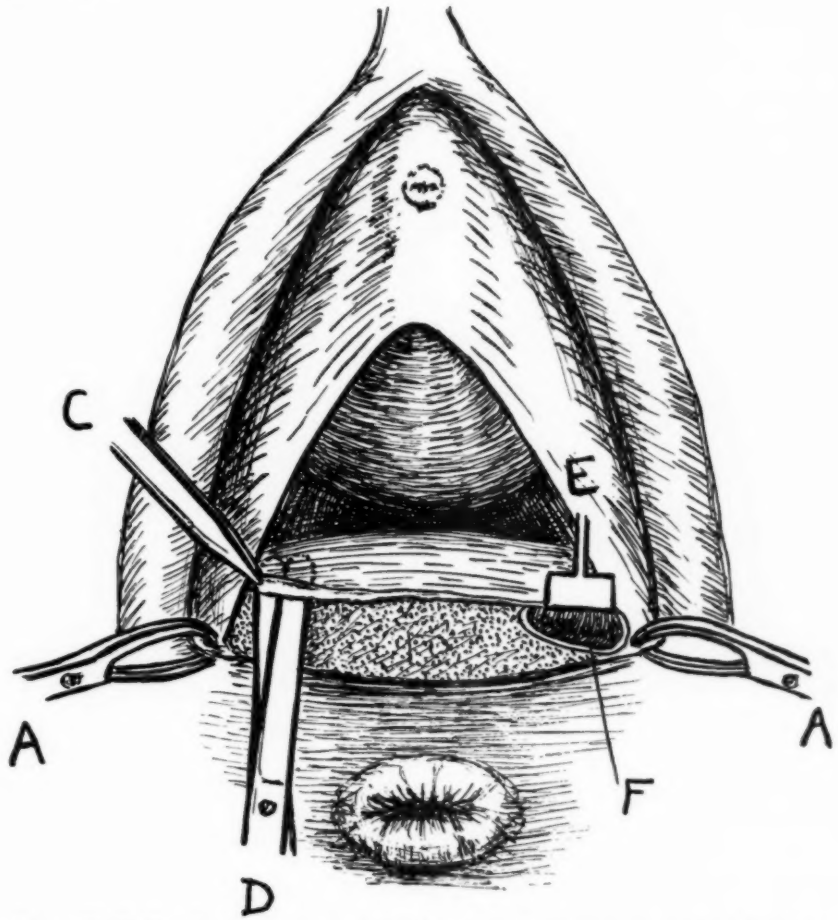


FIG. 166. Bullet forceps A-A at each edge of excised mucocutaneous junction; B-C mouse tooth forceps holding edge of mucous membrane while scissors D are inserted under edge of mucous membrane into plane of cleavage, opened and withdrawn open; edge of separated mucous membrane grasped with "T" clamp E; F, separated plane on left side.

Step II. Separation of the Posterior Vaginal Mucous Membrane. The outer edge of the vaginal mucous membrane is grasped with a mouse-tooth forceps, lifted, and a pair of curved Mayo scissors is introduced under it laterally and upward. With a short snipping motion a line of cleavage is found and the scissors are pushed into this plane opened and removed open,

so as to leave a wide plane of separation. The separated edge of vaginal mucosa is grasped with "T" clamp. The same procedure is carried out on the opposite side. (Fig. 166.) The two

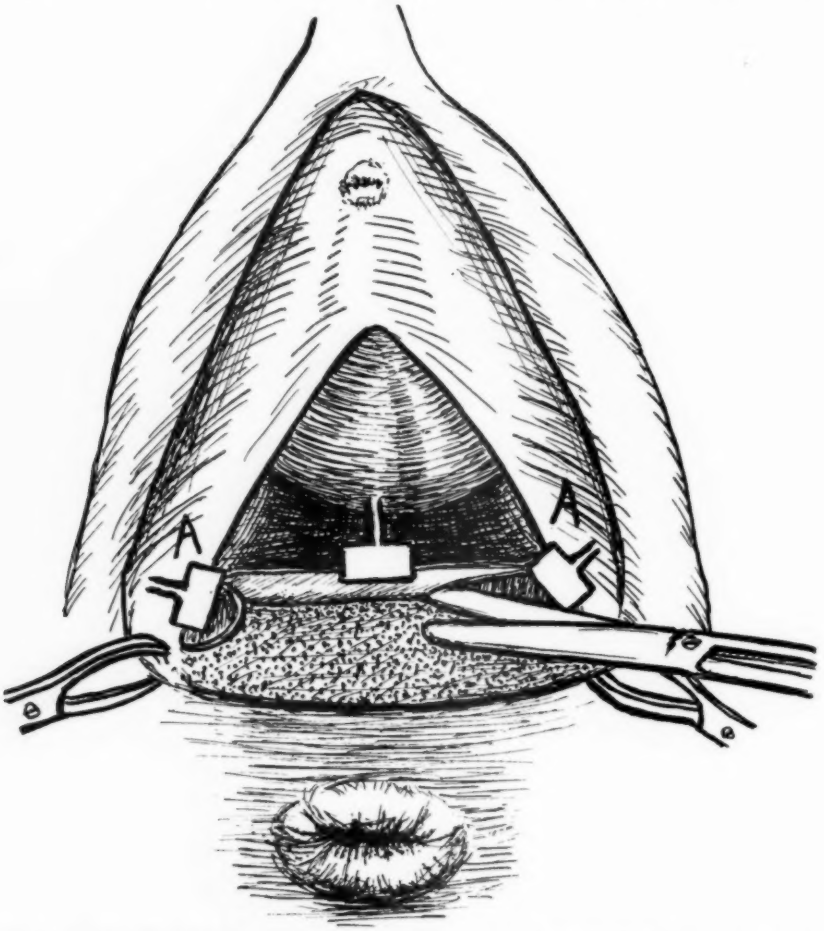


FIG. 167. Two "T" clamps A-A are held taut and the vaginal mucous membrane is separated from rectum with scissors along the plane of cleavage.

"T" clamps are held taut and the vaginal mucosa is separated from the underlying rectum by cutting along the line of cleavage close to the vaginal mucous membrane, from the operator's right to left. (Fig. 167.) The center portion of the vaginal mucosa is grasped with a "T" clamp and the separation of the vaginal mucosa is extended upward and laterally until both levator ani muscles are exposed. If a rectocele or enterocele operation is to be performed, the posterior vaginal mucosa is separated up to the cervix at this time (see Rectocele).

Step III. Suture of Levator Ani Muscle and Fascia. In isolating the levator ani muscles, one must remember the anatomical relationship of these muscles to the vagina. The

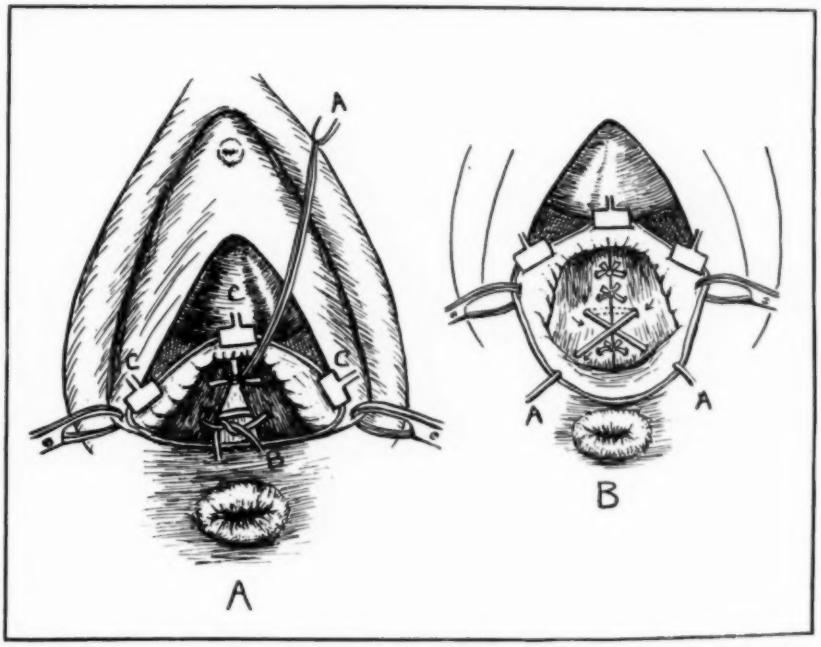


FIG. 168. A. Suture of levator ani muscle to its fellow on the opposite side. A, first suture held long, B, second suture tied, C, "T" clamps on vaginal mucosa. B. Showing levator ani muscle sutured to its fellow on the opposite side with three sutures. A-A figure of 8 stitch from within outward to close superficial tissue.

muscles are in the lateral wall of the vagina close to its outlet. With the index finger in the wound, the operator can feel the levator ani muscle on each side. It is essential to remember that these muscles go from behind forward and act as a sort of sling. With the finger on the upper surface of the left muscle a needle is introduced at right angles to the muscle and through its entire thickness from above downward as far forward as is deemed necessary. The same needle is threaded on the other end of the catgut and it is passed through the right levator ani from above downward. This suture is No. 2 chromic catgut, cut in three. It is tied and the introitus is measured with the fingers. As many of these women are still in active sex life it is essential not to leave too small an opening into the vagina. This suture is held by an assistant with a clamp, thus putting

the levator ani muscles on the stretch. The left index finger is introduced into the cleft between the muscles and thus protects the rectum (Fig. 168) while the levator ani muscles are approxi-

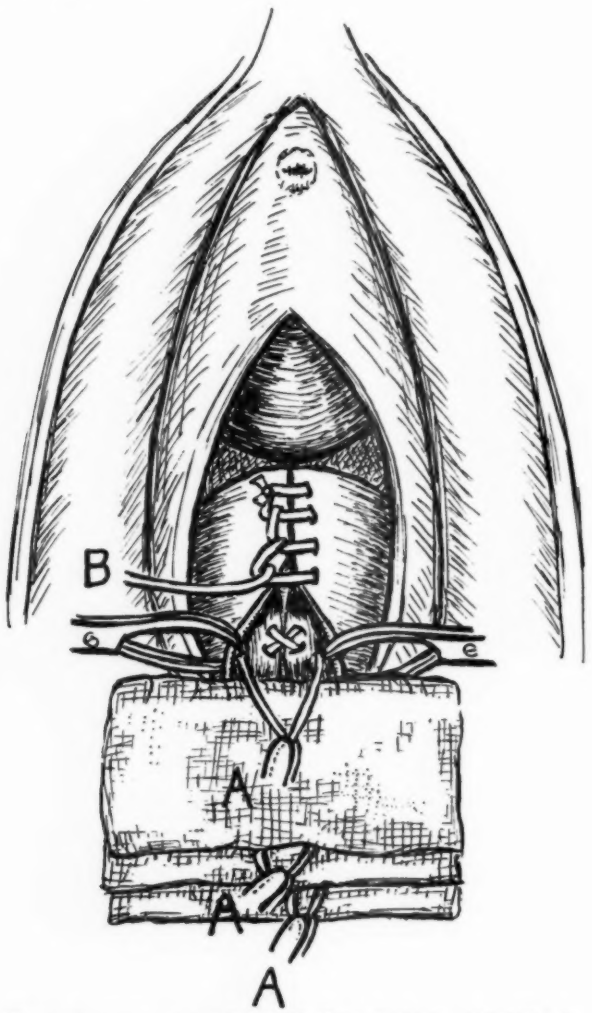


FIG. 169. A-A-A superficial stitches closing perineal body, clamped, held long and separated by sponges. B, lock stitch on cut edge of vaginal mucosa.

mated by two or more sutures, bringing their edges together in front of the rectum.

Caution. Do not place these sutures too close to the lateral pelvic wall. If this is done, when the sutures are tied, the muscles will be torn away from their pelvic wall attachment.

Step IV. Closure of the Superficial Tissue. The tissue superficial to the levator ani muscle is closed with three interrupted sutures of No. 3 chromic catgut, cut in three with a Mayo needle at each end.

The suture catches a small piece of levator ani muscle, so as to obliterate dead space, then takes a good bite in the superficial tissue from within out on the right and then on the left side. These sutures are brought out just subcutaneous to the cut edge of the skin. Three of these sutures are usually necessary to close the space. They are clamped but not tied and are separated by a small sponge between each suture. (Fig. 169.) These sutures are tied after Step v.

Step V. Removal of Excess Vaginal Mucosa and Closure of Vaginal Wall. At the point where the vaginal mucosa crosses the levator ani muscles a suture is passed of No. 3 chromic catgut, cut in two on a Mayo needle, tied and held taut. This marks the upper edge of the vaginal mucosa which is trimmed away from the tenaculi to the suture, removing a triangular piece of tissue. The cut edge of the vagina is then united with a lock stitch. (Fig. 169.) A lock stitch is used here for hemostasis. Following this the three sutures inserted in Step IV are tied and cut long.

A piece of two-inch vaseline gauze packing, 1 yard long, is inserted into the vagina for two purposes:

1. To cause obliteration of dead space by forcing the mucous membrane of the posterior vaginal wall against the rectum.

2. To prevent contamination of the wound until the wound has at least closed itself with serum. The edge of the gauze should come down over the perineal wound.

This packing is removed at the end of twenty-four hours. If the patient can void, she is allowed to do so of her own free will.

LATE REPAIR OF COMPLETE LACERATION OF THE PERINEUM

Preparation of the Patient. Before any surgery can be attempted on a complete laceration of the perineum, the

patient must have a thorough preparation of the gastrointestinal tract as well as the field of operation. This requires five or more days of hospitalization. The vulva is shaved and the vagina and rectum are thoroughly cleansed. The vagina, rectum and surrounding tissues are painted daily with 4 per cent aqueous solution of mercurochrome, and on the day of the operation they are painted three times with Scott's solution (2 per cent mercurochrome, acetone and alcohol).

During this period of preparation the patient must be on a low residue diet, such as egg white, boiled egg, glucose, fruit juices, candies etc.—anything with high caloric value and low residue. The patient's gastrointestinal tract must be thoroughly cleansed. For the first three days she receives very drastic catharsis, magnesium sulphate 1 or 1½ oz., or castor oil 1 oz., every night. On the fourth day she receives an enema and that night, a colonic irrigation. Nothing is done to the rectum on the morning of the operation. As a result of this preparation the patient should be comfortable and not require a bowel movement for several days.

THE SURGICAL PROCEDURE. (WARREN-RISTINE OPERATION)

Operating Room Set-up. As for any vaginal operation. (Fig. 148.)

Step I. Making the Posterior Flap. The patient is placed in the lithotomy position with her buttocks well over the edge of the table. The operative field and a large adjacent area is painted with Scott's solution and draped. An incision is made 1 to 1.5 cm. above the mucocutaneous junction on the posterior rectovaginal septum. This incision is directed outward and downward so that it can be extended to the outer side of the two corrugated dimples which mark the ends of the sphincter ani muscle. (Fig. 170.) This incision extends only through the mucous membrane of the posterior vaginal wall. The vaginal mucosa is dissected downward leaving it attached at its lowermost portion. This apron flap is allowed to hang down in front of the rectum and protect the wound. If the tear has extended upward into the rectum for a short distance, the flap can take

the place of the missing rectal wall. After separating the apron, its edges are attached to a sponge to cover the rectum and the operative area is again painted with Scott's solution. (Fig. 171.)

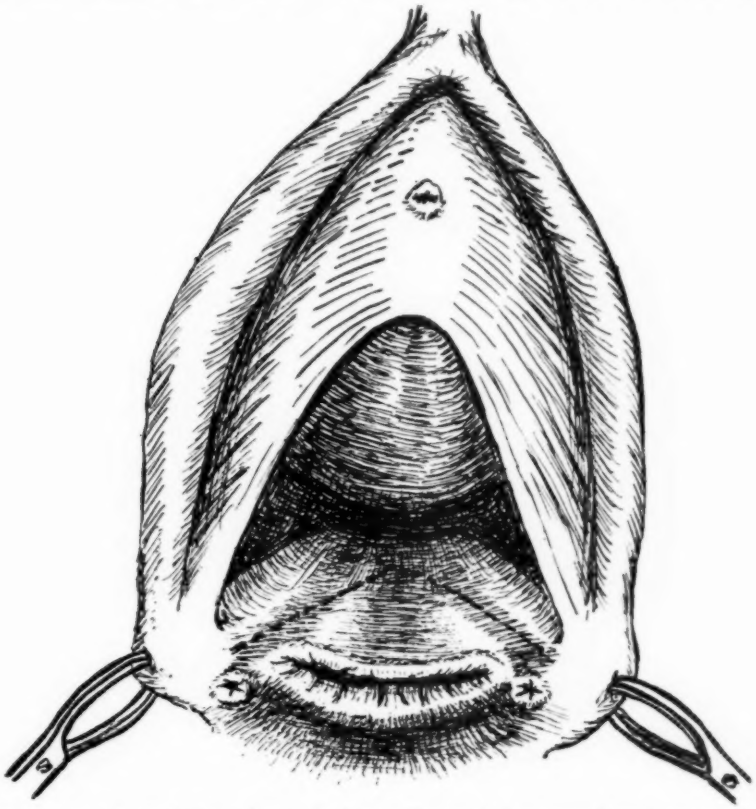


FIG. 170. Dotted line shows incision directed downward and outward towards corrugated dimples marking end of sphincter ani muscle.

Step II. Isolation of Sphincter Ani Muscles. The skin incisions are extended outward about 1 cm. above and to the outer side of the sphincter ani dimples. The skin and subcutaneous tissue is cut and an attempt is made to catch the edge of the sphincter ani muscle. To do this, place an Allis or blunt nosed clamp into the loose areolar tissue directing it downward and outward toward the dimples; when it is over the dimple, the clamp is closed, catching the edge of the sphincter and bringing it into the wound. The same procedure is carried out on the opposite side. (Fig. 171.)

Must the sphincter ani muscle always be caught? One cannot help but feel that this is not of such tremendous impor-

tance. If any muscle tissue in that region is caught it will be either the sphincter ani or a portion of the levator ani muscle. If enough of the levator ani muscle is brought in front of the

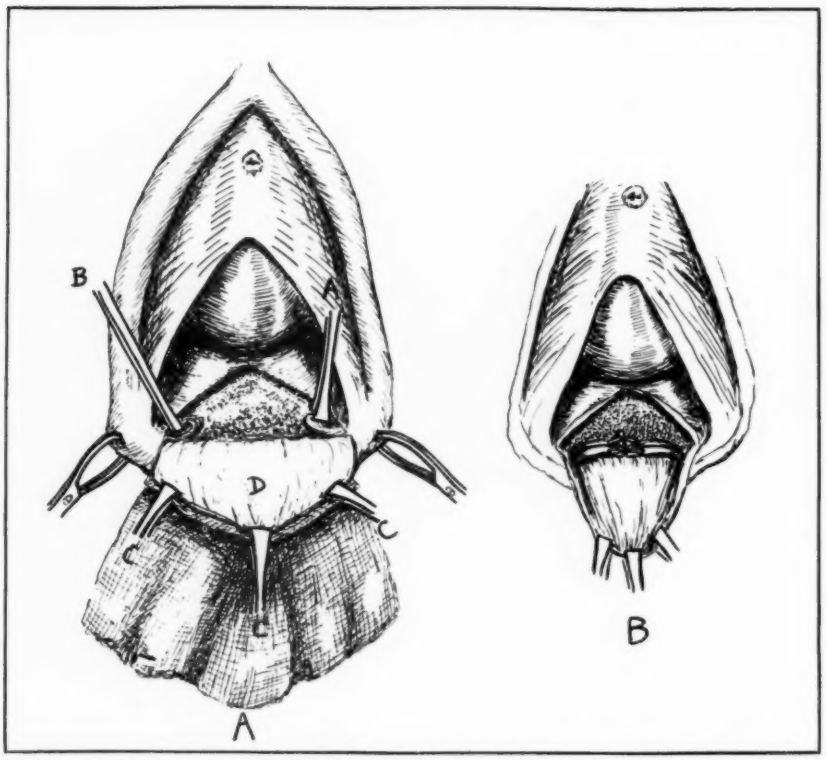


FIG. 171. A. Mucous membrane apron D, turned downward and C-C-C Allis clamps holding gauze sponge—covering anus. A, Allis clamp holding left edge of sphincter ani muscle, B, Allis clamps searching for right edge of muscle. B. Sphincter ani muscle sutured in front of apron.

anal ring and sutured to its fellow on the opposite side, the patient will reestablish a sphincter from the levator ani muscle. All that is actually necessary is to be sure that muscle tissue is brought together in front of the anal ring.

The muscle on one side is sutured to the muscle on the other (Fig. 171, B) with No. 1 chromic catgut, cut in three using two or three sutures. Some men use a nonabsorbable suture for this stitch. If chromic catgut is used for the sphincter stitch, one may augment this with two figure of eight non-

absorbable sutures through the skin and the sphincter ani muscle. As soon as the sphincter ani muscle has been sutured, the rest of the procedure is the same as in an ordinary perineorrhaphy. Before proceding, however, the area is painted with an antiseptic and the gloves are changed.

Step III. Perineorrhaphy. Same as simple perineorrhaphy, beginning at Step II (p. 447).

Postoperative Care of Complete Lacerations of the Perineum. This is very important. Following the operation, the patient is placed on a constipating, low residue diet similar to the one used in the preliminary preparation. She should be given something to eat or drink at frequent intervals, every two or three hours. A mild opiate such as deodorized tincture of opium (paregoric) drams one, every four hours, can be given during this period (six days). On the sixth day a small catheter is introduced into the rectum and 6 oz. of warm olive oil is injected and allowed to be retained. The patient is then given mineral oil by mouth, one tablespoonful every hour, so that within twenty-four hours she will be leaking mineral oil from the rectum. On the seventh day a more drastic cathartic, such as one-half oz. of milk of magnesia is given. When the patient has the urge to move her bowels, a small warm soapsuds enema can be given and she should be instructed not to strain but to allow the bowels to move spontaneously of their own free will, thereby reducing the strain on the wound to a minimum. The bowels must not become constipated. The patient should use mineral oil and milk of magnesia even after leaving the hospital.

Rectocele. Rectocele is the result of the loss or break in the fascial support of the posterior vaginal wall thus allowing a herniation of the anterior rectal wall. This permits the fecal current to go in the direction of the hernia, thus increasing the pouch in the vagina. Patients with large rectocele pouches will frequently complain that in order to have a bowel movement they must insert their fingers in the vagina and change the direction of the fecal current. *A simple perineorrhaphy does not cure a rectocele.*

To cure a rectocele it is necessary to separate the posterior vaginal wall from the rectum up to the cervix and then reunite the ruptured fascial plane in front of the rectum.

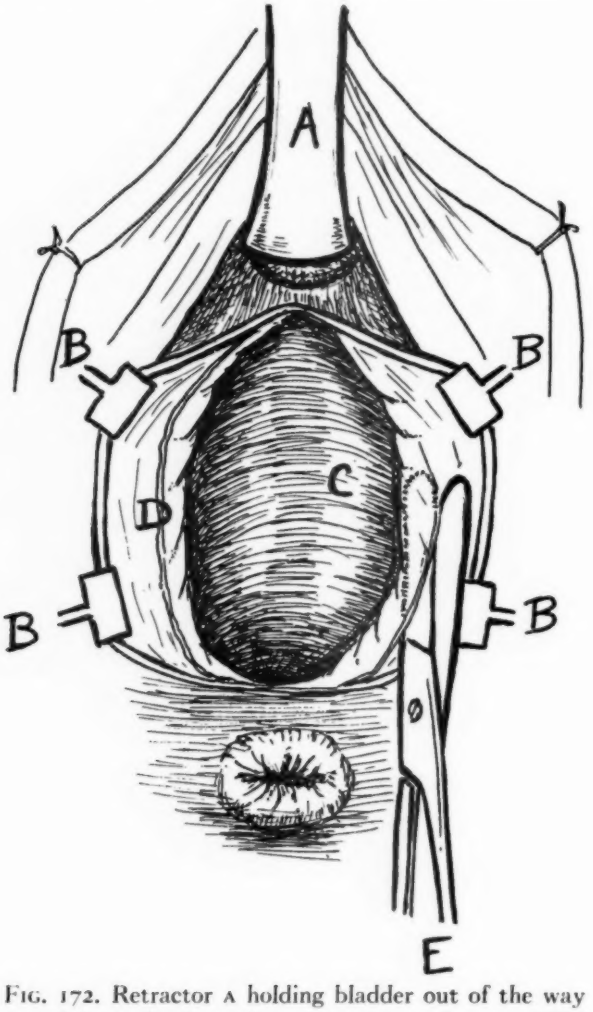


FIG. 172. Retractor A holding bladder out of the way and just allowing edge of cervix to show. B, "T" clamps attached to cut edge of vaginal mucosa, C, bulging rectum, D, cut edge of fascial plane on right side, E, scissors separating edge of fascia from undersurface of vaginal flap.

Operative Procedure. This operation is usually performed in connection with a perineorrhaphy. After Steps I and II of the perineorrhaphy are performed the vaginal mucosa is incised in

the midline and separated from the underlying rectum up to the cervix. The edges of the vaginal mucosa are grasped with "T" clamps, and the mucosa is separated laterally exposing the anterior wall of the rectum. The fascial plane which will be used to close the hernial opening is separated from the vaginal mucosa. This fascia is always found attached to the undersurface of the vaginal mucosa, and can be separated from it along a line of cleavage with straight scissors. (Fig. 172.) This fascial layer is united to its fellow on the opposite side with interrupted stitches of No. 2 chromic catgut, cut in four on a Mayo needle. The union of the fascial layer in front of the rectum closes the opening in the fascia through which the anterior rectal wall has prolapsed and thereby cures the rectocele. The redundant vaginal mucosa is removed, and the cut edge of the vagina from the cervix down to the point where the perineal body will be reconstructed is closed with a continuous lock stitch of No. 2 chromic catgut, cut in two on a Mayo needle.

Caution. Do not remove too much posterior vaginal wall and create a useless test-tube vagina.

Question: Should a woman with a complete laceration of the perineum have a baby?

Answer: If she can become pregnant there should be no objection to her going through another pregnancy without doing a repair of the perineum.

Question: How long should one wait after an unsuccessful repair before attempting another one?

Answer: From nine months to one year.

Question: What kind of suture should be used on the rectal wall?

Answer: Use silk sutures and tie the knots in the rectum. Another layer of tissue should always be placed over this, using a continuous, fine chromic suture so as to invert the rectal wall and bring a broad surface for coaptation. .

Question: Why don't you use any sutures in the skin in a perineal repair?

Answer: It is almost impossible to sterilize this region. Therefore one operates in a partially infected field, and edema and exudate will occur post-operatively. If the skin is sutured with a continuous stitch this serum cannot escape and there is a greater tendency toward infection. Some men go so far as to make numerous punctures in the reconstructed perineal body when marked swelling occurs, to allow the fluid to drain.

[*To be continued in subsequent issues.*]